SURGERY, GYNECOLOGY AND OBSTETRICS

AN INTERNATIONAL MAGAZINE, PUBLISHED MONTHLY

VOLUME L MAY, 1930 NUMBER 5

BONE CHANGES IN HYPERPARATHYROIDISM[1]

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ENLARGEMENT of the parathyroid glands in certain diseases of the skeleton, such as rickets, osteomalacia, and osteitis fibrosa, were noted at necropsy by careful observers more than 20 years ago. Erdheim (10), in 1907, expressed the belief, and offered experimental evidence to support his theory, that these instances of hypertrophy were an attempt on the part of the organism to compensate for the loss of calcium and thus a result rather than a cause of the disease.

While studying a case of osteitis fibrosa cystica, Schlagenhaufer, in 1915, recommended the removal of double palpable tumors of the parathyroid glands. Maresch, one of his associates, concurred in this opinion, but J. Bauer, the surgeon who was responsible for the case, rejected the suggestion as too radical and dangerous a procedure. Mandl, however, was possessed of a more adventurous spirit. In 1925, he gave Erdheim's theory a clinical test. Four parathyroid glands taken from the moribund victim of an accident were successfully transplanted to the abdominal wall of a man aged 38 years with generalized osteitis fibrosa, but the bone disease became definitely worse following the transplantation. Mandl then decided that the previously mentioned hypertrophy of the parathyroid glands in similar skeletal conditions might be a cause and not a result of the bone changes. In an exploratory operation, he found a tumor mass behind the left lobe of the thyroid gland and removed it, and at the same time he removed the parathyroid glands which had been transplanted to the abdomen. The tumor proved to be an adenoma of a parathyroid gland and following its removal there was steady improvement in the condition of the patient.

Since the report of Mandl's case, parathyroid tumors have been removed from several other patients suffering from similar skeletal diseases. The similarity between the symptoms and observations in some of these cases, especially that reported by Barr, Bulger, and Dixon, at St. Louis, and that of Wilder at the Mayo Clinic, and the case which we shall report here, led us to make a diagnosis of hyperparathyroidism.

Miss L. J., aged 59 years, came to the University of Chicago Clinics in May, 1929, complaining of pains in the bones of the legs, particularly in the feet, bowing of both legs, and general weakness. Although she had noted the bowing for only 2½ years, the pains and weakness had been present for 3 to 5 years. The most recent development had been pain in the left hip. The weakness was so marked that she had fallen many times, and on such occasions she could not regain her feet without assistance.

In spite of the fact that she had been breast fed as an infant the patient had suffered from infancy and until she was 3 or 4 years of age with an almost constant diarrhœa. She was never given cod liver oil and after she was weaned she had refused to drink milk. She first walked when 3 years of age. Dental caries developed early and while still a young woman all of her teeth were extracted. While she was never strong and never seemed to feel well, the only definite illnesses which she could recall were rheumatic fever

[1]This work has been conducted under a grant from the Douglas Smith Foundation for Medical Research of the University of Chicago.

TABLE I. ANALYSES

Date	Blood serum				Product of Ca & P
	Calcium	Phosphorus	CO_2 Content	pH	
1929	Mg. per cent	Mg. per cent	Vol. per cent		
May 28	12.55	2.81	64.2	7.46	35.6
June 6	11.40	3.38	64.9	7.54	39.5
June 10	12.17	3.72	64.2	7.57	45.2
June 13	11.93	2.93	63.7	7.53	35.0
June 24, operation					
June 25	9.39	2.53	64.8	7.48	26.1
June 26	8.33	5.10	63.5	7.49	43.0
June 27	6.97	4.91	63.3	7.48	34.3
	Symptoms of tetany.				
June 27*					
July 1	6.98	5.76	63.4	7.47	40.7
July 7	8.26	4.76	65.0		39.4
July 9	8.60	4.98		7.45	42.8

*Calcium lactate 6 grams daily given June 27 to 30.

TABLE II.—METABOLISM EXPERIMENT, JUNE 3 TO JUNE 9, 1929*

	Daily averages				
	Intake	Output		Total	Balance
		Urine	Fæces		
Calcium	Gm. 0.292	Gm. 0.311	Gm. 0.168	Gm. 0.479	Gm. −0.187
Phosphorus	0.799	0.498	0.207	0.705	+0.094
Nitrogen	10.920	8.69	0.830	9.520	+1.400

*The diet during this period was held constant. Aliquot portions (1/10) of all food were set aside, dried, and thoroughly mixed for analyses. Indigocarmine was used to mark the fæces of the period. We are indebted to Dr. Callie Mae Coons for these analyses.

at the age of 9 and typhoid fever at the age of 18 years. Cardiac weakness, with decompensation, had caused dyspnœa on exertion and some palpitation, and this dated back to the attack of rheumatic fever.

The examination revealed a fairly well developed and fairly well nourished woman who did not look as old as her stated age (Figs. 1 and 2). One was struck by the lack of tissue turgor, for her skin, the subcutaneous tissues, and the muscles themselves seemed to hang limply and in folds, giving the impression of extreme hypotonicity. All reflexes were present and equal but very sluggish.

The cardiac area was moderately increased in size and a loud systolic murmur was heard precardially and in the axillary line. A spherical nodule 2 centimeters in diameter could be palpated in the lower pole of the right lobe of the thyroid gland. Skeletal deformity was noted as follows: a marked kyphosis of the thoracic spine, slight thickening of both femora, and some thickening and slight inward bowing of both tibiæ and fibulæ.

In walking there was just the slightest tendency to waddle, and genu valgum was noted. This "knock knee" deformity was not at all obvious when the patient was lying down, but increased upon standing. In addition there was eversion of the feet, and this became so marked after standing for a few minutes that the medial malleoli were less than an inch from the floor and the pes planus which was barely noticeable when she would first stand up became quite marked.

The body weight was 54.6 kilograms and height was 169 centimeters. The pulse rate was 75 per minute. The temperature was normal, the blood pressure 134-90, and the basal metabolic rate was plus 4. The erythrocytes numbered 4,000,000.

Roentgenograms revealed osteoporosis of the calvaria, osteoporosis and bowing of the femora, and the same rarefaction of the pelvic bones and the lumbar vertebræ with sinking in of, and male-like, pelvis (Figs. 3, 4, 5, and 6). Particularly noticeable was the thinness of the cortices of the shafts of the long bones. No sign of bone cyst or tumor was present.

Serum calcium was slightly elevated, assuming 9 milligrams to 11 milligrams for each 100 cubic centimeters of serum to be the normal range, while the serum phosphorus was lower than normal (Table I). Metabolic studies from June 3 to June 9 revealed a negative calcium balance and showed the excretion of an abnormally large proportion of the total calcium output by way of the kidneys (Table II).

A diagnosis of hyperparathyroidism was then made on the basis of the syndrome consisting of: pain and bowing in the weight-bearing extremities; osteoporosis of the bones of the skeleton; progressive muscular weakness; elevated serum calcium and lowered serum phosphorus; a palpable nodule in the lower pole of the right lobe of the thyroid gland; and a negative calcium balance. Since it was thought that the nodule in the right lobe of the thyroid gland was a parathyroid tumor, exploration for the tumor was advised.

The operation was performed by Dr. D. B. Phemister on June 24, 1929. The right lobe of the thyroid gland was found to be about double the normal size and there was a nodular enlargement in the body of the lower pole. This nodule when excised proved to be a thyroid adenoma and not a parathyroid. The surfaces of the upper and lower poles were searched for enlarged parathyroids and none was found. The left thyroid lobe was about normal in size and a nodule projected from the posterior inferior portion. This had a pedicle containing blood vessels. It measured about 1 by 1¾ centimeters and was removed. A small mass near the upper pole, considered to be a normal parathyroid, was also removed.

The postoperative course was normal until June 26, 2 days after the operation, when the patient was

TABLE III.—METABOLISM EXPERIMENT, JULY 1 TO JULY 6*

	Daily averages				
	Intake	Output		Total	Balance
		Urine	Fæces		
	Gm.	Gm.	Gm.	Gm.	Gm.
Calcium	0.303	0.058	0.389	0.447	—0.144
Phosphorus	0.784	0.450	0.375	0.825	—0.041
Nitrogen	8.51	5.760	1.66	7.42	+1.090

*The diet during this period was held constant and as nearly like that given in the previous experiment as possible. Aliquot portions (1/10) of all food were set aside, dried, and thoroughly mixed for analyses. Indigocarmine was used to mark the fæces of the period. We are indebted to Dr. Callie Mae Coons for these analyses.

TABLE IV

Date	Blood serum				Product of Ca & P
	Calcium	Phosphorus	CO_2 Content	pH	
1929	Mg. per cent	Mg. per cent	Vol. per cent		
Aug. 19	11.11	3.64	61.2	7.63	40.4
Aug. 20	Lost	3.904	66.4	7.57	
Aug. 22	Lost	4.35		7.46	
Aug. 26	10.57	4.212	65.4		44.5
Aug. 28	11.05	3.8	68.3	7.52	42.0

hyperirritable and hypercritical, anxious, moving restlessly in bed, and complaining of tingling fingers and numbness of the hands. No definite Chvostek sign could be elicited, but the tendon reflexes were all exaggerated. On the following morning, as the serum calcium was found to be at what is usually considered to be a tetany level, 6.97 milligrams for each 100 cubic centimeters of serum, the administration of calcium lactate (2 grams three times daily) was begun. The restlessness quickly became less marked and the serum calcium began to rise to higher values. The serum phosphorus rose to normal values immediately after operation (Table I).

A metabolic balance, run for the 6-day period July 1 to July 6, revealed a decided change in the mode of excretion of calcium (Table III). Before operation nearly 65 per cent of the calcium had been eliminated by way of the kidneys, while the renal fraction after operation was reduced to about 11 per cent of the total, the fæcal fraction being correspondingly greater. The negative calcium balance of this period may be accounted for in part by the fact that, contrary to orders that all intake be limited to the diet and distilled water, the patient was given mineral oil for the first 3 days of the experiment. Gross particles of food and much oil were noted in the fæces and this fact suggests that absorption of food including calcium was somewhat interfered with. A further and probably more important source of error is the fact that until 24 hours before the beginning of the experiment, the patient had been receiving 6 grams of calcium lactate daily. While an indigocarmine marker was used it is possible that the first sample of fæces contained some of the calcium given as medicine and not previously expelled. The low calcium content of the urine is ample evidence that the calcium in the blood and tissues was being retained. The diet in this metabolic period was identical with that used in the pre-operative metabolic period, June 3 to June 9, both being weighed diets of pre-determined composition.

The patient returned to the clinic on August 21, stating that she was much stronger and was having less pain, although following a fall she had noticed some pain in the left thigh. The observations at the general physical examination were essentially like those at the previous admission, with the additional notation that the patient seemed to be more alert mentally, reflexes were more brisk, and she was generally more active than at the time of the first admission. The blood count was higher, 5,100,000 erythrocytes and 80 per cent hæmoglobin (Dare), and the basal metabolic rate was plus 1.

The appearance of the bones roentgenologically showed no change that could be detected at this admission, which was less than 2 months after the operation. The serum calcium, while not so high as when she first came to the clinic, was at the upper limits of normal, while the serum phosphorus during the admission was also a good normal, ranging from 3.6 to 4.3 milligrams (Table IV).

On November 12, 1929, the patient stated that she still had some pain in both tibiæ after standing for several hours, but she declared that her strength was good and that, in contrast to her previous lassitude, it was difficult for her to reconcile herself to a daily rest period. She had gained 7 kilograms in weight since leaving the hospital following the operation. Reflexes at this visit were active. Serum calcium was 10.22 milligrams and serum phosphorus 4.21 milligrams. Calcium excretion in the urine for a 24 hour period, with patient on her routine diet, was 206.57 milligrams. This marked increase in urinary calcium output suggests a return toward a negative calcium balance but is not above the upper limit of normal. The general tone of the muscles was obviously much better than before operation, but no change in the degree of skeletal deformity could be determined.

An X-ray examination on December 12, 1929, showed no change in the calcium content of the bones. The patient was given viosterol (irradiated ergosterol) with instructions to take 5 drops three times each day.

She was seen again in the clinic on January 3, 1930, and was feeling exceedingly well at that time, and stated that she was stronger than she had been in more than 10 years. Prior to the removal of the parathyroid tumor she had been unable to remain on her feet for more than a very short time because of weakness. For more than 3 years before coming into this clinic she had been so weak that she often fell down, apparently from the fact that her knees

Fig. 1 (left). The patient. There is obvious loss of tissue turgor and muscle tone. (Before operation.)

Fig. 2. The lower extremities. Note the lateral curvature of the femurs, the genu valgum, the medial curvature of the bones of the legs, the eversion of the feet, and the marked flattening of the plantar arches of the feet.

simply gave way under her, and she would not be able to regain her feet unassisted. She estimates that these falls occurred on the average of about two times each week. Since the operation 5 months ago she has fallen only one time and that was soon after leaving the hospital and was occasioned by stumbling. The pain in her feet which had almost completely incapacitated her for any kind of work for 2 or 3 years prior to her admission to this hospital has not been present since leaving here nearly 5 months ago. She does have some pain in the distal third of the tibiæ which comes on when she has been on her feet for an hour or longer, but this does not interfere with her daily routine. Serum calcium at this visit was 10.92 milligrams and phosphorus was 4.82 milligrams for each 100 cubic centimeters of serum. These figures are well within the range of normal.

The last visit to the clinic was on March 10, 1930. Some aching of the tibiæ had persisted, but her strength was good. Serum calcium was 12.57 milligrams and phosphorus 5,712 milligrams for each 100 cubic centimeters of serum.

Throughout the course of these studies the hydrogen ion concentration and carbon dioxide content of the blood serum remained fairly constant, although both were slightly higher than the accepted normal (Tables I and IV).

Pathological studies were made of the tissues removed at operation. The three specimens consisted of a wedge shaped mass of nodular thyroid tissue, 2 centimeters in diameter, from the lower pole of the right thyroid gland, a small nodule from the upper pole of the left gland, and the tumor.

The wedge shaped mass of nodular thyroid tissue was not particularly interesting. Microscopic studies showed that except for the relatively large number of colloid containing vesicles, in a few of which the lining epithelium was slightly flattened, this section did not vary greatly from normal (Fig. 7). The lining epithelium was smooth and regular and no portion of the section seemed to consist of an actively secreting gland. There were several areas of dense lymphocytic infiltration.

The second specimen, which proved to be a normal parathyroid gland, was a firm, ovoid, pinkish nodule, measuring 2 by 4 millimeters, with a delicate connective tissue capsule. The section stained with hæmatoxylin and eosin was seen to be composed almost entirely of cells resembling epithelial cells with a connective tissue network which divided the cells into irregular strands (Figs. 8 and 9). These epithelial cells were of two types. By far the most abundant were relatively small cells with large nuclei containing many dark staining granules. There were also a few groups of large cells with relatively clear cytoplasm which stained pink with the eosin. In the central portion of the section there was an occasional vesicle or acinus. Numerous fat cells were noted and the entire section was quite vascular.

The tumor mass removed from the vicinity of the lower pole of the left thyroid gland was a smooth, reddish brown, semi-elastic, meaty nodule 1 by 1¾ centimeters, with a fibrous capsule and a definite vascular pedicle (Fig. 10). Microscopic sections stained with hæmatoxylin and eosin revealed a compact cell structure composed of cells resembling epithelium cells with numerous acini or alveoli which were lined with cuboid or low columnar epithelium, in a number of which colloid material was noted (Figs. 11 and 12). There were numerous blood vessels and while the bulk of the gland was composed of the alveoli, in some portions of the section, particularly near the periphery, the epithelial cells were arranged in strands with blood spaces and some connective tissue between. No keratotic figures were found, but the nuclei of most of the cells were filled with large dark staining granules. A very few of the large clear cells noted in the normal gland were seen. As in the case reported by Wilder, no foam cells or fat cells were present.

The structure of the tumor differed from that of the normal parathyroid gland in: the presence of numerous acini; the compactness of the cellular elements; the absence of fat cells; the absence of any strand-like or trabecular arrangement of the epithelial cells; the preponderance of deeply staining granular cells; and the scarcity of the large cells with clear, pink staining cytoplasm. The patho-

logical diagnosis was benign adenoma of the parathyroid gland.

Procedure in serving calcium balance diet. The following diet was planned and the procedure in serving it was supervised by Miss Florence Smith, in charge of the dietetic service of the hospital: protein 49 grams per kilogram body weight; fat 82 grams per kilogram body weight; carbohydrate 156 grams per kilogram body weight; calories 1556 (basal plus 15 per cent). Estimated calcium content 0.300 grams.

These constant diets were served on 6 consecutive days, breakfast on the first morning being served after indigocarmine was given and after venous puncture. All dishes in which food was served and silver used on tray were washed, dried, and then rinsed in distilled water. Dishes in which hot food was served were placed in food truck to warm. All foods served on trays were weighed on balance scales and placed in dishes in which they were to be served, then one-tenth that amount weighed for laboratory sample. Chemically clean beakers with covers were secured from the laboratory each morning and labelled with the patient's name, room number, and date on both beaker and cover. The total day's allowance of butter was weighed first. One-tenth of the total was placed in a beaker for analysis and allowed to stand in hot water until the butter melted, then the beaker was tilted and turned until the butter was well distributed on the walls of the containers. This procedure prevents food sticking and it is more easily removed from the container after drying. The day's allowance of bread, sugar, fruits, and cereal for breakfast was weighed and one-tenth portion of each food was placed in the beaker. All cooked foods were weighed hot, and dishes in which they were to be served were placed in a food truck to maintain constant temperature. All food was cooked with distilled water. The eggs served were cooked in the shell, removed from the shell with a spoon which had been rinsed in distilled water, and then weighed in the cup in which they were served. On the sixth day raw eggs were beaten in a bowl which had been rinsed in distilled water, and an amount equal to one-tenth the total weight of all eggs served was placed in the beaker containing the food aliquots for analysis.

On the fourth day of the second balance study period the patient was given approximately ¾ glass of orangeade by error of the nourishment nurse. To minimize the error 15 grams of orangeade were added to the laboratory specimen on the sixth day. On the next day the patient was given carmine in the morning before the regular diet was resumed.

Methods of chemical analysis. Food composites collected as described were left on a steam bath until practically dry, then removed to an electric oven at 80 degrees C., further dried to constant weight, and then ground and mixed for analyses.

The fæces, without transferring, were covered with acidified alcohol and dried in the same manner as the food. All collections for the period were composited and ground for analyses.

Fig. 3. The calvaria showing osteoporosis. May 28, 1929.

Urine was collected into bottles containing toluol. One-fifth of the total excretions for each 24 hours were combined and preserved with toluol and strong hydrochloric acid until analyzed.

Analyses for calcium were carried out according to the McCrudden method, the hydrogen ion concentration being adjusted with sodium acetate, as described by Shohl. The precipitated calcium oxalate was collected on a Gooch crucible, ignited, and weighed as calcium monoxide. The determinations were made on triplicate samples of food and fæces after they had been ashed in an electric muffle furnace and the ash dissolved in hydrochloric acid. The acidified filtered urine was evaporated and ashed and dissolved in hydrochloric acid.

The procedure used for phosphorus analyses involved moist oxidation with sulphuric and nitric acids, according to Neumann, and then double precipitation, first as ammonium phosphomolybdate, finally as magnesium ammonium phosphate, following the McCandless-Burton technique. The last precipitate was collected on a Gooch crucible, ignited, and weighed as magnesium pyrophosphate.

For total nitrogen determinations the Arnold-Gunning (21) modification of the macro-Kjeldahl procedure was used.

The calcium in the blood serum was determined by the Clark and Collip modification of the Tisdall and Kramer method. Phosphorus in the blood serum was determined by a slight modification of Fiske and Subbarrow. Carbon dioxide content was determined by the Van Slyke method and the hydrogen ion concentration was estimated by the colorimetric method as described by Hastings and Sendroy, the Hasting's bicolorimeter being used.

Fig. 4. The proximal ends of the femurs and the pubic and ischial bones showing decalcification and thinning of the cortices. May 28, 1929.

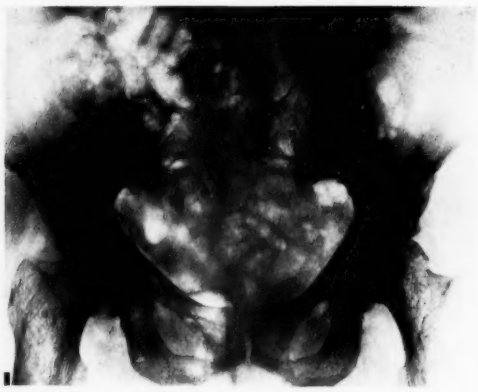

Fig. 5. The pelvis and lower lumbar spine showing decalcification. May 28, 1929.

Including the case of Mandl which was reported in 1926, there are descriptions of 8 cases, similar to that here reported, from which tumors of one or more parathyroid glands have been removed. In seven of these cases, those of Mandl, Gold, Barr, Wilder, Boyd, Snapper, and finally in our own case, a varying amount of improvement followed extirpation of the tumor. It is further learned at the time of going to press with this paper that another case of Barr has been operated on and an adenoma of one of the parathyroid glands removed with beneficial results. Wilder stated that following the removal of the parathyroid tumor in the case which he reported, the patient noted marked improvement in strength and in muscle tone and relief from pain in the bones, and roentgenologically there was some increased density of the bones and disappearance of a tumor of the maxilla. Mandl very recently reported that 3 years after the operation the condition of his patient is still favorable. Before removing the adenoma of the parathyroid gland the patient had been bedridden for months, but she now has no pain and is able to take long walks with the aid of a cane.

In the case of Beck, reported in 1928, and diagnosed as generalized osteodystrophia. An olive sized tumor was removed from the lower pole of the right thyroid gland and a coffee bean sized tumor from the site of the upper parathyroid gland on the same side. The patient developed tetany on the fifth day after and died 20 days after the operation. At autopsy no parathyroids could be found on the left side.

Diagnoses of adenoma of parathyroid glands as an explanation of a condition considered to be due to hyperfunction of the tumorous glands may not be accepted without some criticism. In discussing adenomatous changes in the thyroid gland, Rienhoff (26) declared that even though the cells lining the alveoli do function locally, there is no proof that these neoplasms produce a toxic secretion, and there is no evidence to suggest that these cells lining the alveoli function in such a manner as to affect the organism as a whole. Aschoff did not believe that adenomata of the parathyroid glands had anything to do with the bone changes occasionally found associated with them. Rienhoff (28) also states that adenoma and carcinoma of the parathyroid glands have in some instances been associated with low blood calcium and even with tetany.

Pathologically the tumors in all of the cases reported have been diagnosed adenoma. Functionally they resemble true hyperplasia. Both Ewing and Harbitz have recognized the difficulty of differentiating between a moderate diffuse hyperplasia and a true adenoma of the parathyroid gland. Tumors of the parathyroid gland grossly similar to those reported in this series, and associated with similar skeletal conditions, were studied by

Erdheim and diagnosed hyperplasia. Strauch studied a tumor which was removed from the neck of a woman who died after a typical attack of puerperal osteomalacia. His diagnosis of hyperplasia was based upon the presence of all of the normal cell elements while, according to his belief, only one type of cell is found in the true adenoma.

The tumor which was removed from our patient was almost entirely composed of the one type of large epithelial cell with hyperchromatic nuclei and the predominating characteristic was one of acini formation. No portion of the gland resembled normal parathyroid tissue. We feel justified, then, from a pathological standpoint, in making a diagnosis of parathyroid adenoma. If this were a compensatory hyperplasia as Erdheim and others believe, it is difficult to explain why another gland on the same side should be entirely normal. If we then assume that the changes in the calcium metabolism were due to increased parathyroid activity, we must conclude that either this adenoma was producing an abnormal amount of parathyroid secretion; grossly normal parathyroid glands were hyperfunctioning; or that there was some undiscovered and abnormally active accessory parathyroid tissue. The presence of a pathologically similar tumor in a rapidly growing series of reported cases lends weight to the first hypothesis.

The tumors of Wilder and Mandl were classified as malignant adenoma because of the presence of mitotic figures, the polymorphism of the cells, the hyperchromatic nuclei, and in Wilder's case because of the invasion of the neoplastic tissue into the capsule. The striking absence of foam cells and of fat which Wellbrock mentions may not be a criterion of malignancy, for the same thing was true in our case and other cases in which the pathological study was reported in detail and in which there was no suggestion of malignancy.

The duration of the bone disease in both Wilder's and Mandl's cases of from 5 to 7 years speaks against a diagnosis of malignant tumor. However, a review of the literature of malignant tumors of the thyroid and of the parathyroid glands indicates that in almost every instance tumor of some kind preceded

Fig. 6. The tibiæ and fibulæ showing osteoporosis, thinning of the cortices, and inward bowing. May 28, 1929.

the malignant changes for several years. Balfour, in a series of sixty-three cases of malignant struma, found that in not a single case had the condition appeared suddenly, but some form of diffuse or nodular goiter had preceded it. Wilson found that in 157 of 290 cases of malignant goiters, there had been an enlargement for 5 years or longer. In most of the 8 cases of malignant tumors of the parathyroid gland reviewed by T. Kocher, the tumor had been present for many years. Metastases to the neck, mediastinum, or pleura occurred in each of the cases described by Kocher.

In the case of Guy, the tumor of the neck had been noted for 5 years. When removed the section showed a few acini and a few mitotic figures with areas of degeneration and cyst formation resembling those found in Wilder's tumor. A diagnosis of adenoma of the parathyroid gland was made, but the patient returned after 11 months with 3 new nodules near the operative scar. The author noted here that her general health was good and there was no evidence of skeletal disease. These nodules and several glands in the posterior cervical triangle of the neck were removed and all were found to be carcinomatous and all recurred in spite of X-ray therapy. Guy concluded from his studies that apparently benign tumors of years' duration may suddenly take on malignant characteristics. While it is doubtful that the malignant changes in the tumor were responsible for bone changes which have been noted in so many other cases in which there was no suggestion

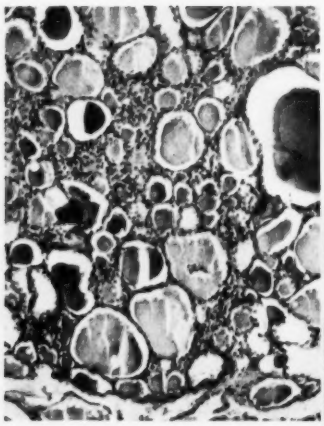

Fig. 7.

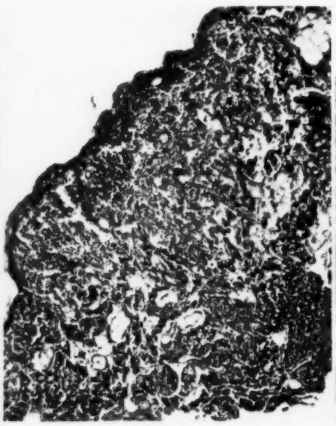

Fig. 8.

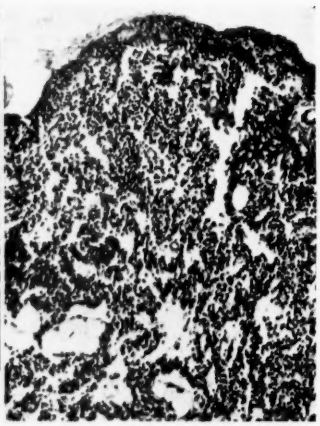

Fig. 9.

Fig. 7. Section taken from the thyroid gland showing the large colloid filled vesicles, with smooth lining epithelium. (× 150.)

Fig. 8. The normal parathyroid gland. The cells which resemble epithelial cells are loosely arranged in trabeculæ. There are a number of fat cells. (× 70.)

Fig. 9. The normal parathyroid gland showing an occasional acinus. (× 150.)

of carcinoma, the cases in the literature of malignant tumors of parathyroid glands help to substantiate the pathological diagnoses of malignancy in the cases of Wilder and of Mandl.

Richardson, Aub, and Bauer explored the neck of a patient of DuBois with the clinical picture of hyperparathyroidism and after finding no tumor, removed two normal appearing parathyroid glands. The improvement in the condition of the patient which followed this operation was so marked that it indicated that hyperparathyroidism may result from hyperfunction of otherwise normal glands.

Bergman, of Berlin, reported a similar case in which, following studies leading to a diagnosis of generalized osteodystrophia fibrosa, the surgeon was not able to find any tumor, but did identify four normal parathyroid glands, which he was afraid to disturb.

Duken, of Berlin, basing his conclusion upon the clinical picture adds 2 more cases to the list of those diagnosed as hyperparathyroidism. His patients were the youngest of the series, 1 being 7 years of age and the other 14. A diagnosis of late rickets had been made, but from the X-ray and metabolic studies, the author made the additional diagnosis of osteodystrophia fibrosa and expressed the belief that the condition was probably due to derangement of function or to tumors of the parathyroid glands. Palpable tumors similar to those which in other cases had proved to be parathyroid adenomata were noted in the older of his two patients, but neither case had been subjected to an operation at the time of his report, and his diagnosis had thus not been confirmed. A definite diagnosis by palpation cannot be established between an adenoma projecting from the thyroid and a parathyroid tumor, and the presence of a palpable tumor is not essential for the diagnosis as shown by our case.

Intensive metabolic studies in a case of osteomalacia, with the effects of treatment over a period of 1 year, have recently been reported by Blumgart and his associates. Marked improvement was noted when the patient was given a diet that was rich in vitamin D. When cod liver oil concentrate and ultra-violet light were given with adequate amounts of calcium and phosphorus, there was calcification of the softened bones and disappearance of all symptoms. On the basis of their results in this case the authors conclude that in their patient osteomalacia was definitely a vitamin-deficiency disease.

A review of the symptoms of the patients reported by different writers, together with the findings at examination, reveals a number

Fig. 10.

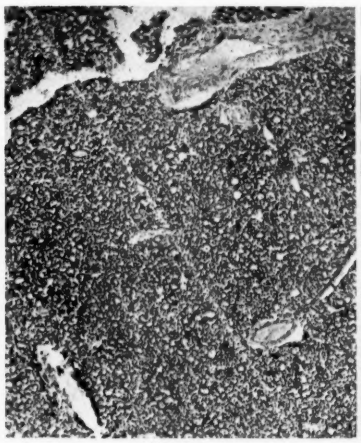

Fig. 11.

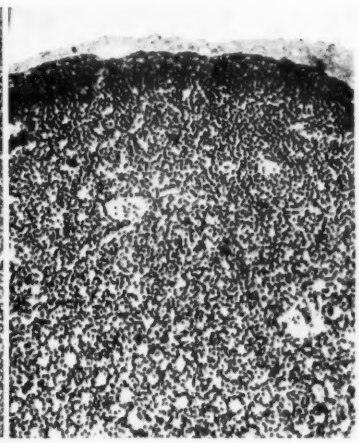

Fig. 12.

Fig. 10. Gross appearance of tumor. Tissue has been removed from the side of the specimen for microscopic study.

Fig. 11. The tumor. The cell structure is compact and there are numerous alveoli or vesicle-like spaces. (× 70.)

Fig. 12. The vesicles of the tumor are lined with cuboid epithelium and in some of them colloid-like material is seen. (× 150.)

of similar conditions. All of the patients in which a case history is given complained of progressive muscular weakness, pain and bowing of the weight-bearing extremities, and general lassitude. In all of the patients there was osteoporosis of the bones of the skeleton. In the cases in which blood chemistry studies were made there was an elevation of the serum calcium which varied from slightly above the upper limits of normal to the extremely high figure of 23.60 milligrams for each 100 cubic centimeters of serum which was reported by Snapper (Table V). In the 5 cases in which calcium metabolism studies were made, there was a negative balance in each case and in each of these cases the balance became positive following operation, except in our own case, for which result an explanation has been offered.

Table V, besides illustrating some of the points mentioned of similarity between the various cases reported, shows that there were 7 females and 4 males, while Bergman does not mention the sex of his patient. Fractures had occurred in 5 of the cases. In the cases of Gold, Barr, and Beck, the fractures which had occurred during the course of the disease healed very slowly, but in Snapper's case the fractured femur united firmly soon after operation. An ununited fracture of the femur brought Beck's case to him and symptoms and findings leading to his diagnosis of osteitis fibrosa were brought out during the subsequent examination. The death of this patient on the twenty-first day after operation precluded any conclusions regarding the healing of the fracture after removal of the parathyroid adenoma. Tetany was noted after operation in 3 cases. In all cases in which the chemistry of the urine was studied there was found a calciuria and, following removal of the tumor, the calcium of the urine fell to below normal values.

Complete descriptions of the roentgenological studies were not included in most of the case reports. In all of the 12 cases there was diminished bone density, spoken of by some as generalized osteoporosis and by others as decalcification. In 6 cases, in addition to the generalized loss of density of the bones, there were cysts of the femurs or of the pelvic bones. These were noted in the cases of Gold, Richardson, Barr, Duken, Wilder, and Snapper. Following removal of the parathyroid tumor, Mandl, Richardson, Barr, Wilder, and Snapper reported X-ray evidence of improvement, as shown by increased density in calcium content of the bones. No positive

TABLE V.—RÉSUMÉ OF CASES REPORTED

Case	Age	Diagnosis	Bone cysts reported	Giant cell tumors of bones	Fractures	Tetany after operation	Tumor of parathyroid
1. Mandl	48 Male	Osteitis fibrosa					Adenoma.
2. Beck	41 Female	Osteitis fibrosa		Amputation of right leg for sarcoma 3 years before	Left femur	Died in tetany 21 days after operation	Adenoma.
3. Bergman	Not given	Osteitis fibrosa					No tumor found.
4. Gold	54 Female	Osteitis fibrosa	Yes		Left femur poor union		Adenoma.
5. Richardson, Aub, and Bauer	34 Male	Osteitis fibrosa	Yes		Several	None	No tumor. Two normal glands removed.
6. Barr, Bulger, and Dixon	56 Female	Hyperparathyroidism	Yes	Present, healed after operation	Right clavicle Left humerus Healed slowly	Yes, severe, 3 or 4 days after operation	Adenoma.
7. Duken, Case 1	7 Female	Late rickets and osteodystrophia fibrosa					No operation.
8. Duken, Case 2	14 Female	Late rickets and osteodystrophia fibrosa	Yes				No operation. Bilateral palpable tumors.
9. Boyd, Milgram, and Stearns	21 Male	Osteomalacia or osteitis fibrosa				Chvostek pos. No tetany	Cystic adenoma.
10. Wilder	32 Female	Osteitis fibrosa	Yes	Healing followed removal of tumor			Malignant adenoma.
11. Snapper	56 Male	Generalized osteitis fibrosa cystica	Yes		Right femur and several others Healed after op.	Positive Chvostek and pos. Trousseau's. Cramp of hands and carpopedal spasm	Adenoma.
12. Our case	59 Female	Osteomalacia				Slight twitching	Adenoma.

evidence of healing of the bone cysts was included in these reports. In the case of Boyd and his associates, no roentgenological evidence of improvement was noted 2 months after removal of the parathyroid tumor. In our own case the X-ray appearance of the bones remained unchanged more than 7 months after removal of the tumor.

Giant cell tumors of the bones were noted in the cases of Wilder and of Barr, and these were found to be healed within a few months after removal of the parathyroid tumor. In the case of Beck the right leg had been amputated, following a diagnosis of sarcoma, 3 years before the parathyroid tumor was found. When we consider the presence of giant cell tumors in the cases of Wilder and of Barr, we are inclined to suggest the possibility that the lesion of the right leg of the case of Beck, which did not recur following amputation, may have been a benign giant cell tumor also.

Based largely upon the X-ray appearance of the bones (Table VI), the diagnosis in 7 of the 12 cases, those of Mandl, Beck, Bergman, Gold, Richardson, Wilder, and Snapper, was generalized osteitis fibrosa, or osteitis fibrosa cystica in the cases in which bone cysts were noted. Barr and his associates avoided the issue and made a diagnosis of hyperparathyroidism. Duken classified his 2 cases as late rickets and osteodystrophia fibrosa. Boyd and his associates were undecided whether to call the condition osteomalacia or osteitis fibrosa. Because of the normal appearance of the bone trabeculæ, the generalized loss of the calcium salts from the bones, the absence of cyst-like areas, the slight bending of the bones of the weight-bearing extremities, and the sinking in of and male type of pelvis, we have diagnosed

TABLE VI.—RÉSUMÉ OF CASES REPORTED

Case	X-ray		Blood studies. Mgm. Ca. and P. per 100 c.cm. serum				Mgm. Ca. excretion in urine for 24 hrs.		Symptomatic improvement
	Before operation	After operation	Before operation Ca.	P.	After operation		Before op.	After op.	
1	Generalized osteoporosis	Improvement	None made				54 mgm.	7.6 mgm. 11 days after	Marked. Gained 16 kgm. in weight in 2½ years.
2	Generalized osteoporosis	Patient died in 21 days							Patient died.
3	Generalized osteoporosis	No operation							
4	Generalized osteoporosis and cysts	Not reported	13.1		Ca. 9.9 after 27 days		412 mgm.	26.4 mgm. 5 days after	Marked. Had gained 11 kgm. 6 mo. after operation.
5	Generalized osteoporosis and cysts	Marked increase in ca. deposits of bones, 2 yrs. later	13.1 to 15.3 mgm.	1.4 to 3.2 mgm.	No appreciable change		6 to 7 times greater than in normal controls	No change reported	Marked.
6	Generalized osteoporosis and cysts	Improvement	16.1	1.4	Tetany after operation				Marked.
7	Generalized osteoporosis	No operation	14 mgm.	5 mgm.					No operation.
8	Generalized osteoporosis and cysts	No operation	20.75 mgm.	3 mgm.					No operation.
9	Generalized osteoporosis	No improvement noted in 2 mo.	17.6 mgm.	2.2 to 3.3					Yes.
10	Generalized osteoporosis and cysts	Improvement Inc. density of skeleton	11.64 to 12.79	1.71 to 2.11	Ca. 9.06 to 7.13	P. 1.71 to 1.99	220 to 340	14 mgm. (av.) 1 week after	Marked.
11	Generalized osteoporosis and cysts	Marked improvement	19.0 to 23.6		Ca. 7.2 to 9.04		322 to 411 mgm.	2.2 to 48 mgm.	Yes.
12	Generalized osteoporosis	No improvement in 5 mo.	11.5 to 12.55	2.81 to 3.72	Ca. 6.97 after 2 days to 11.1 after 2 mo.	P. 4.76 to 5.76	311 (av.)	58 (av.) during first two weeks after	Yes. Gained 7½ kgm. within 4 mo. after operation.

Metabolic studies showed negative balance in cases of Wilder, DuBois, et al, Barr, Boyd, et al, and in our case. These studies were not carried out or were not reported in the other cases. In all except our case the balance became positive immediately after removing the tumor.

the skeletal condition present in our case as osteomalacia.

The rather striking similarity between this entity which Barr and his associates have called hyperparathyroidism and rickets is pointed out by Wilder. Treatment with ultra-violet light and a diet rich in vitamin D resulted in marked gain in strength and in weight in his case, improvement of anæmia, and retention of calcium and phosphorus. He suggested that a rôle of vitamin D is the inhibition of the activity of the parathyroid glands. This hypothesis is given additional support by the reports of Starlinger and Blumgart of great improvement in cases of osteomalacia through administration of irradiated ergosterol or of diets rich in vitamin D and ultra-violet light therapy. Likewise, Weil has noticed an improvement of patients suffering from osteodystrophia fibrosa after irradiation of the parathyroids.

SUMMARY

A case of osteomalacia is reported in which a diagnosis of hyperparathyroidism and tumor of the parathyroid gland was made and confirmed at operation. A second parathyroid was excised and found to be normal.

A résumé of 11 other cases which have points of similarity and which have been recently described is briefly discussed.

Symptomatic improvement was noted in our case following removal of the parathyroid adenoma.

While removal of the tumors of the parathyroid glands has not resulted in complete recovery in reported cases, the symptomatic improvement, the chemical evidence of increased calcium retention, and the X-ray evidence of increased density of the bones in a few cases followed for a long enough period after operation are results which, as the operative risk is slight, do warrant surgical intervention in cases such as that reported here.

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POSTOPERATIVE PULMONARY COMPLICATIONS AND BRONCHIAL OBSTRUCTION[1]

Postoperative Bronchitis, Atelectasis (Apneumatosis) and Pneumonitis, Considered as Phases of the Same Syndrome

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IT is the aim of this paper to show the very close relation between the postoperative pulmonary complications usually described as postoperative bronchitis, atelectasis, and pneumonia; furthermore to show that these conditions generally follow one another in the order named without clear cut distinctive signs and that they represent evolutional phases of one and the same postoperative pathological process—bronchial obstruction.

This theory, if correct, is of far more than theoretic interest. It is of practical importance because it is only with exact knowledge of the etiology and interrelation of these postoperative complications that we shall be able to establish a rational curative treatment based not on the symptoms but on the causes, and furthermore that we shall be able to introduce efficient prophylactic measures.

The study of massive atelectasis, so thoroughly carried out in recent years gives, I believe, the key to the solution of the important problem of postoperative pulmonary complications—factors of such prime importance to the surgeon.

In his commendable papers, Elwyn (46) gives his opinion on the etiology of postoperative pneumonia, which in essence is as follows: There is first a partial or total collapse of the lung; if the affected part does not expand within 24 to 48 hours pneumonia may develop, depending merely upon the presence or absence of bronchial inflammation and upon the extent and severity of such inflammation. But, he concludes: "This explanation does not entirely solve the problem; it merely puts it back a step further. The question is, how does the collapse of the lung arise? At present we have no answer." It is exactly the purpose of this paper to endeavor to answer this question.

In previous studies on atelectasis and experimental and human lobar pneumonia, Coryllos and Birnbaum (27, 28, 29) came to the conclusion that pneumococcus lobar pneumonia is a pneumococcic lobar atelectasis. In support of this view were presented experimental and clinical evidence to show that both conditions are accidents in the course of pneumococcic bronchitis and are due to the obstruction of bronchi by bronchial secretions or exudate. Differences in clinical evolution depend upon the presence, type, and virulence of the micro-organisms present in the occluding secretion or exudate.

Further study of postoperative complications offered new evidence in favor of this theory. Briefly stated, I believe that because of the stagnation in the bronchial tree of bronchial secretions or exudate after operation, a bronchial occlusion may ensue and lead to atelectasis. The size of the obstructed bronchus determines the anatomical distribution of the disease—whether it will be multilobar, lobar, or lobular. The outstanding factor in the production of these complications would, therefore, be *bronchial occlusion and suppression of the free drainage of the pulmonary airways* by means of which normally the lung is maintained in an aseptic condition even though a great number of micro-organisms are introduced with the inspired air. When bronchial obstruction is once established, the type of complication will depend upon the microbes present in the bronchial exudate; aerobes and anaerobes as pneumococcus, streptococcus, staphylococcus, influenza bacillus, spirochætæ, fusiform, bacillus perfringens, etc.—all normally or accidentally present in the upper respiratory tract may play a part. I certainly do not exclude the possibility of postoperative pulmonary complications secondary to large or

[1]From the Laboratory of Surgical Research, Cornell Medical College and the Second Surgical Division of Bellevue Hospital (Cornell). Received for publication September, 1929.

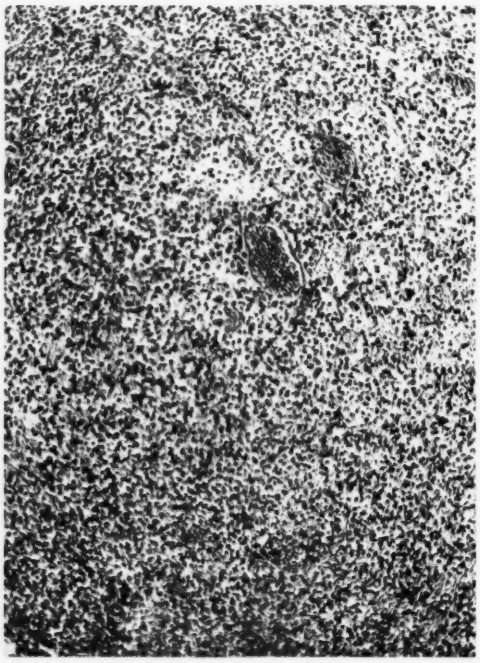

Fig. 1. Dog 195. Photomicrograph of experimental obstructive atelectasis. Simple, uncomplicated apneumatosis. Notice the dilated vessels. Compare with Figure 7.

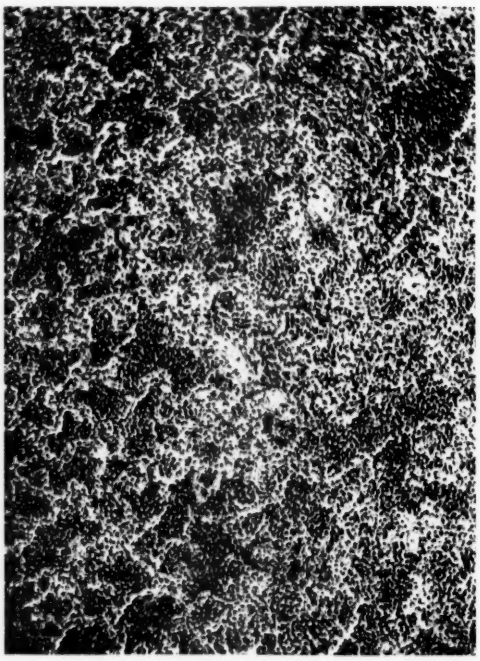

Fig. 2. Dog 199. Experimental obstructive massive atelectasis. Complicated by pneumonitis. Dilated vessels are easily distinguished. Compare with Figure 9.

small emboli. But, I believe with Wharton and Pierson (139) that embolism can and should be differentiated from the "inflammatory lesions" with which I am dealing.

Under different headings are to be considered: First, the etiology of postoperative atelectasis and of postoperative pneumonitis; second, a comparative study of their similarities; third, a discussion of the theory proposed; and lastly, an outline of a new prophylactic and curative measures.

ETIOLOGY OF POSTOPERATIVE MASSIVE ATELECTASIS

Massive atelectasis can no longer be considered as a rare postoperative complication. I completely agree with the opinions of Mastics, Spittler and McNamee, and Lee, Clerf, and Tucker, that the incidence of this complication is not as low as has been stated: 0.6 per cent by Scott and Cutler, 0.8 per cent by Pasteur (99), and 1.3 per cent by Scrimger, but that it is in its different forms, lobular, lobar or massive, closer to 50 to 70 per cent. I only want to add that a great number of evanescent forms of atelectasis are often undiagnosed when they do not give rise to marked clinical symptoms, or are diagnosed as "congestion" or "hypostasis" of the lung.

In a previous paper (29) the different theories for the etiology of this condition were extensively discussed and experimental and clinical evidence was given in favor of the theory of mechanical obstruction of a bronchus by mucous exudate.

This theory, first confirmed experimentally by Lichtheim in 1879 and supported by Elliott and Dingley, was given clinical proof by the work of Jackson and Lee, Tucker, Clerf, Harrington, Hearn, and others, who by repeated bronchoscopic examinations not only verified the occlusion, but, by aspirating the occluding mucus, produced rapid inflation of the diseased lung and cured the

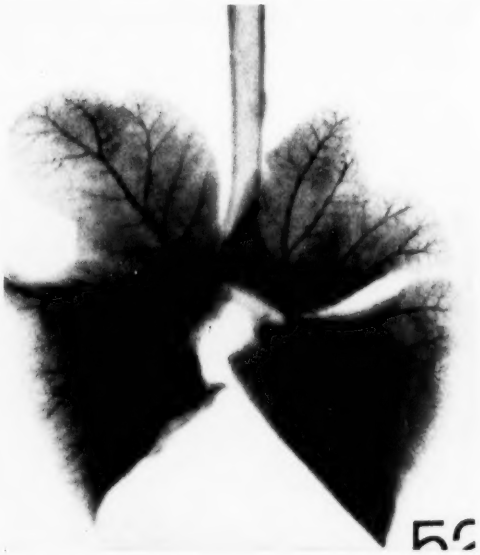

Fig. 3. Dog B 52. Obstructive atelectasis of middle and inferior right lobes. The obstructing balloon is filled with sodium bromide and it is visible in the right common bronchus. The pulmonary vessels are injected with lipiodol, the injection being made into the jugular vein on the living animal. The branches of the pulmonary artery are perfectly injected. There is no difference whatever between the vessels of the healthy (left lung and upper right lobe) and the atelectatic portions of the lung. (Roentgenogram of the lungs extracted from the chest.)

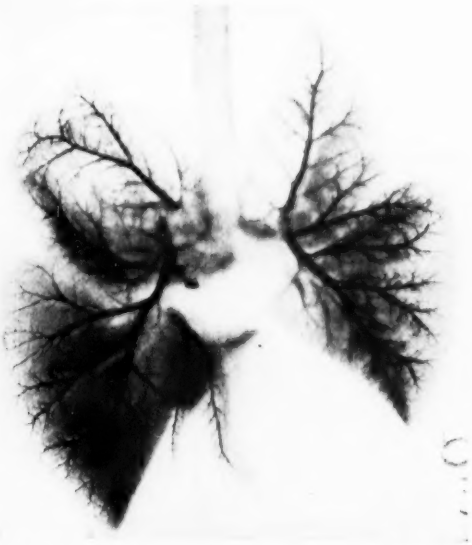

Fig. 4. Dog B 57. Experimental pneumonia of the right lower and subcardiac lobes. (Twenty-four hours after insufflation through the bronchoscope into the right common bronchus of 10 cubic centimeters of pneumococcus type one culture.) Lipiodol injection on the living animal; same technique as in Figure 3. There is no difference between the healthy and consolidated lobes. (Roentgenogram of the lungs extracted from the chest.)

atelectasis. In a recent paper, Lee, Clerf, and Tucker have given a practical demonstration in support of this theory: They cured postoperative atelectasis by bronchoscopic aspiration of the mucous exudate and produced a typical atelectasis in the anæsthetized dog by introducing into its right bronchus the material which was aspirated from the patient.

Although it is almost universally conceded that a complete occlusion of a bronchus produces an atelectasis because of subsequent absorption of alveolar air, there is much discussion about the mechanism of the obstruction in postoperative atelectasis. A clear conception of this phase of the problem will go a long way toward solving its difficulties.

In the main, two theories are supported. One is the "nervous reflex theory" and the other the "mechanical occlusion of a bronchus by bronchial secretions." I firmly believe that the key to the solution of the whole question of postoperative pulmonary complications lies in a clear understanding of these theories, and it will be well worth our effort to discuss their merits fully.

Fig. 5. Dog B 76. Roentgenogram of the heart of an atelectatic dog, injected with lipiodol through the jugular vein. (Same technique as Figures 3 and 4.) The right heart is filled up with oil, but none passed into the left heart.

NERVOUS REFLEX THEORIES

There is no single nervous reflex theory; different authors describe different reflexes originating at different points, transmitted by different pathways, and producing the same result in different ways. They can be classified as: (1) the diaphragmatic, (2) the bronchoconstrictor, (3) the vasomotor. I shall give a résumé of each of them and then discuss them.

1. *Diaphragmatic and muscular theory.* W. Pasteur considered atelectasis secondary to paralysis of the diaphragm, whereas Briscoe considered it secondary "to a disturbance of the functions of the diaphragm and associated respiratory muscles due to inflammation affecting the retroperitoneal portion of the diaphragm." Soltau and Soltau and Alexander, and Watson and Meighan believed that a reflex paralysis of the diaphragm was brought about by afferent impulses being conveyed to the respiratory center from the focus of irritation by way of the vagus, the efferent impulses being conveyed by way of the phrenic nerve. Ball considers diaphragmatic fixation as a possible cause of atelectasis in his case of suppurative pancreatitis with an occluded foramen of Winslow. Bradford suggested spastic contraction of the respiratory muscles as a factor. L. Sante believes that several factors existing simultaneously are necessary for the production of the condition, such as inhibition of cough reflex by some toxic reflex stimulus in connection with an impairment of the respiratory function and immobilization of the respiratory muscles from a defense reaction or paralysis from toxic neuritis. This in time permits accumulation of secretion blocking the bronchi and results in atelectasis.

2. *Bronchoconstrictor theory.* The principal defender of this theory is Churchill, who in his exhaustive paper concludes that atelectasis is due to a combination of weakened respiratory force and bronchoconstriction. Experimental support of this theory is presented in the work of Dixon and Brodie (16, 17, 36) who enclosed the lung in an oncometer and were able to produce bronchoconstriction or dilatation either by direct vagal stimulation or by means of injected drugs. Under these conditions they produced either distention or collapse of the lung by varying the force of inflation and the time allowed for deflation. With rapid forceful artificial respiration which allowed only a short time for expiration, bronchoconstriction commonly produced distention. With forceful artificial respiration and intervals sufficiently long to allow full expirations, constriction of the bronchioli resulted in collapse. They found that a lobe collapsed in such a manner, usually remained so, even after bronchial constriction had passed off. Churchill compares the condition of postoperative patients to that of experimental animals of Dixon and Brodie (36) because in the former shortened inspiration and prolonged expiration are present. It is to be noted, however, that Churchill fully admits the possibility of bronchial obstruction by inflammatory exudate or even normal secretion.

Scott and Joelson consider that atelectasis is generally bilateral and is due to bilateral reflex. "In both lungs," they say, "the lumina of the finer air passages are undergoing variation in size resulting from alterations of bronchomotor or vasomotor tonus." "But," they add, "the initial reflex is possibly vasodilator in character, and the most striking feature is an extreme pulmonary congestion, almost an angiomatous condition." In order to explain the transformation from the initial bilateral to a subsequent unilateral atelectasis, they suggest that possibly the obstruction becomes complete on the dependent side because "of greater congestion of the dilated pulmonary capillaries and of a *compensatory hyperventilation* of the other side which keeps those respiratory passages open. Should such hyperventilation fail to take place bilateral instead of unilateral atelectasis is produced." The above is a combined bronchomotor and vasomotor theory. L. Sante (119) expressed a similar opinion in 1927: ". . . the cause is not known, but, it seems most probable that some infection or insult to the region of the vagus supply produces a reflex on the bronchioles, permitting their temporary collapse."

As against the reflex bronchoconstrictor theory, Scafer calls attention to the fact that

vagal section can lead to a collapse of the smaller bronchi from *paralysis* of their muscular layer.

Moore objects to Scafer's opinion on the ground that if it were correct, section of the vagus should result in a decrease of tidal air in the corresponding lung. Moore's experiments showed exactly the opposite: "Immediately following a right vagotomy the tidal air of the right lung rose to 55 cubic centimeters (from 44) and of the left lung to 72 cubic centimeters (from 56 cubic centimeters), and later to 100 cubic centimeters on the right and 138 cubic centimeters on the left."

3. *Vasomotor reflex theory.* Gwyn, in 1923, suggested vasomotor disturbance of the pulmonary circulation as a possible cause of atelectasis. Scott and Cutler, after stating that many etiological factors have been proposed but that no primary cause has been found, express their belief that the "process is initiated by a nervous reflex, probably largely vasomotor, which results in a narrowing of the lumina of the peripheral bronchioles by venous engorgement, swelling of the mucous membrane, and the elaboration of a tenacious secretion." Among the important factors contributing to the completion, the extent, and the localization of the complication, they consider the quantitive changes in ventilation. They believed the diminished ventilation to be not a result but a cause of atelectasis. Lee, in 1924, admitted as possible causes, besides obstruction of the corresponding bronchus, "possibly some paralysis or bronchial spasm due to a reflex irritation from other parts in the body. In his more recent paper (Lee with Tucker and Clerf, 1928), he modifies this opinion and considers that at least the most important factors in the production of apneumatosis are "viscid bronchial secretion and some inhibition of coughing."

Fontaine, Lenormant and Iselin, more recently upheld the same opinion. "Up to this time," says Fontaine, "atelectasis has been experimentally produced only by bronchial obstruction. It seems, however, that the pulmonary nerves have a preponderant part in the production of this symptom in the human. . . . If the direct irritation of the nervous fibres is responsible for massive atelectasis it must act through bronchospasm or reflex vasomotor influence." But he admits that "So far, no definite data have been produced in favor of this hypothesis." Rouillard (1929) does not express any definite opinion although he considers as possible reflex motor disturbances and congestion of the atelectatic lung which could explain the greater opacity of this lung as compared with the simple collapsed lung in pneumothorax. Bowen (1929) in a painstaking paper, containing the most complete historical review of the question, adheres unreservedly to the occlusion theory.

H. Santee and Bergamini and Shepard reported two rapidly fatal cases of bilateral atelectasis and suggested as the cause a vasomotor reflex or an angioneurotic œdema, because of the rapid development of the disease, the engorgement of the capillaries, and the absence of bronchial occlusion at autopsy.

Fontaine and Hermann reported the results of experimental extirpation of all the extrinsic nerves to one lung in the dog. It is interesting that of the ten dogs used in their experimental work, atelectasis occurred suddenly in only one of them on the third day after operation. No bronchial occlusion was found at autopsy; they conclude that "if there were any reflex responsible for this collapse the impulse must have come by way of the anastomotic branches from the opposite side, or they must have originated in the peripheral ganglion of the affected lung." It will be interesting before closing this brief résumé of the different reflex theories to report the experimental findings of Einthoven. After section of the vagi he did not notice the slightest modification in the intrapulmonary pressure or any noticeable changes in the cross section of the bronchioles. He concluded that when the bronchial muscles are at rest, the vagi exert little or no tonic effect upon them.

COMMENT ON THE NERVOUS REFLEX THEORIES

The theories attributing atelectasis to *paralysis of the diaphragm* (reflex or organic)

or to its fixation, are no longer tenable. It is generally admitted that these conditions cannot produce massive atelectasis. In cases of fixed deviation of the diaphragm (as in the case of Ball), we might perhaps have small atelectatic areas, as with pneumothorax or pleural exudate from compression of the lung. But this is totally different from massive atelectasis. It has been demonstrated that from phrenicotomy alone in humans or animals, atelectasis does not occur; and furthermore that the elevation of the diaphragm is not the cause of the atelectasis but the effect of it. Coryllos and Birnbaum (28) showed that if in dogs with phrenic nerve sectioned on one side atelectasis by bronchial obstruction is produced on the other side, the diaphragm corresponding to the atelectatic lung will rise and even to a higher level than the paralyzed one.

I think for these reasons that the theory of diaphragmatic origin of the atelectasis should be definitely discarded, at least as a primary and determining cause.

The reflex bronchoconstrictor theory. As a primary cause of massive atelectasis this theory has no clinical facts in its support. We know, for example, of no case in which reflex irritation of the nasal mucosa has produced atelectasis; whereas this irritation often causes inflation of the lungs and asthmatic attacks. Dixon and Brodie, on whose experimental work Churchill bases this hypothesis, state that a reflex bronchial constriction is experimentally obtained by exciting the nasal mucous membrane, and that little or no result from stimulating the central vagus, superior laryngeal, or corneal nerves is obtained. These same authors state that atropine produces paralysis of the bronchial muscles and dilatation of the bronchi. This fact is of importance in connection with the case of postoperative atelectasis reported by Scott (Case 38 of this author) in which 2.78 milligrams (almost 1/20 grain) of atropine was administered within 30 minutes in 3 doses, while the patient was under fluoroscopic examination and atelectasis persisted. Not only was there no decrease in the density of the lung or the displacement of the heart, but on the contrary, rather a slight increase was noticed. Adrenalin did not have any effect either, although Dixon and Ranson have proved that adrenalin produces an active bronchial dilatation especially marked when an increased tonus of the bronchi is present; the same striking effect should have been noticed in atelectasis, if this condition were due to bronchoconstriction,—as occurs in cases of asthma; nor can the view be sustained that bronchoconstriction starts the atelectasis and the bronchioles are subsequently completely obstructed with mucous exudate, as was suggested by Scott and Joelson, because in that case why should the bronchioles of one particular lobe only be affected and not of the other lobes? Why is there a characteristic lobar distribution of the disease if spastic contraction or occlusion of the small bronchioles is the cause?

Only a mechanical occlusion of a bigger bronchus supplying the whole lobe can explain the lobar distribution of the disease, and a large bronchus cannot be constricted by a reflex or otherwise. Starling (131, p. 897) states that "under the influence of vagal stimulation, or inhalation of carbon dioxide, expiration and not inspiration will be rendered more difficult, because of the different mechanical conditions of the bronchi during the two phases of respiration. . . . Normally the elastic structure of the lung is pulling upon the bronchial wall, tending to maintain it patent and so opposes the action of the bronchial muscles. During inspiration this expanding force is so increased that in the presence of bronchial constriction the ingress of the air is rendered easier the more powerful is the contraction of the inspiratory muscles. On expiration, on the contrary, all parts of the lung collapse, drawn in by the chest wall. The pull of the lung tissue on the bronchial wall is lessened, but is still present. If, however, the respiratory muscles contract vigorously the intrapleural pressure becomes positive and the pull of the lung tissue on the bronchial walls is changed into a pressure tending to obliterate their lumen and so impeding the outflow of air." This physiological mechanism is fully justified by clinical facts. In hypersensitization of the parasympathetic system (vagotonia) with spastic

contraction of the bronchial muscles there is no atelectasis but on the contrary an emphysema. In allergic asthma, in anaphylatic shock of the lung in the guinea pig, and in reflex asthma due to irritation of the nasal mucosa, there is always emphysema and not atelectasis. Dixon and Brodie believed that both constrictor and dilator fibers in the vagus supply the lung on the same side only, and only very few crossed fibers exist. Moore, however, proved that cutting one vagus produces a response from both lungs. How, then, shall we explain by a bronchoconstrictor reflex the cases of bilateral or contralateral atelectasis, if we accept the first view? How will the cases of H. Santee, Bergamini and Shepard (bilateral atelectasis), and the case of the dog with denervated lung of Fontaine and Hermann be explained? Again, if Moore's view is correct, why is the disease lobar and not diffuse and patchy in distribution, as it obviously is in cases of emphysema due to bronchoconstriction? For these reasons I believe that bronchoconstriction cannot and should not be considered as a primary cause of massive atelectasis.

3. *The vasomotor reflex.* The last variety of nervous reflex to be discussed has neither clinical nor experimental facts in its support. There is a mere supposition based upon the pathological findings of distended vessels in microscopic sections, upon the two cases of Bergamini and Shepard in which complete bilateral collapse developed while the patient was still on the operating table and upon the cases in which no bronchial obstruction was found at postmortem examination. At first it would seem that the cases of Bergamini and Shepard could not be explained by bronchial obstruction with mucus even if such obstruction were found, because complete absorption of the air by the alveolar blood cannot be completed in such a short time; in fact, it is known that, although absorption of oxygen and carbon dioxide is completed in a very short time, nitrogen requires 10 to 20 hours. This difficulty, however, is only apparent. My explanation of the two cases of H. Santee and of Bergamini and Shepard is as follows:[1] These patients were under deep gas-oxygen-ether narcosis and breathed an air saturated with ether and nitrous oxide; consequently the rapidity of completion of atelectasis in these two cases would depend upon the absorption coefficient of the gases present in the alveolar air. Teschendorf (1924), studying the time of absorption of different gases in the pleural cavity (where the gases are absorbed by the alveolar capillaries less rapidly than when introduced into the lung), has found that carbon dioxide is so rapidly absorbed that it is impossible to produce a pneumothorax with even 600 or 700 cubic centimeters introduced into the pleural cavity of man. The absorption coefficient of carbon dioxide at 0 degrees C. is 1.7967; of ethylene gas, 0.0946; and of nitrous oxide, 1.3052. The absorption of ethylene gas requires a few minutes, and the absorption of carbon dioxide and nitrous oxide is instantaneous. Van Mechelen in his paper "Ether Narcosis" writes: "The diffusion of ether in the blood of the lung capillaries is so rapid that within 2 seconds 95 per cent of even a massive dose of ether is absorbed." After one single inspiration of 500 to 800 cubic centimeters of air containing 39 per cent ether, only 0.2 gram are found in the expired air; the proportion of the absorbed ether is in direct proportion of its concentration in the inhaled air and in the anæsthetic mixture the quantity of nitrogen is negligible. These facts give the explanation of the almost instantaneous absorption of the anæsthetic mixture in case of bronchial obstruction and of the rapid development of atelectasis, as in the cases of Bergamini and Shepard (7) and the case of Lilienthal. The only remaining argument is that no obstructing plug was found at autopsy. However, it will be shown later that a real "plug" is not necessary to occlude a bronchus and produce atelectasis, but that even a thin secretion may cause this condition if the means of defense of the lung are sufficiently lowered.

So far as the atelectatic dog of Fontaine and Hermann is concerned, I can hardly believe that it presented true atelectasis. The interpretation of the findings in this animal is really puzzling. By a left thoracic incision

[1] I had the opportunity of discussing the above cases personally with Dr. Bergamini who accepted the explanation given here.

the entire vagosympathetic supply of the left lung was carefully excised and a piece of the left lung was removed for microscopic study and the pulmonary wound ligated; this fact is not mentioned in the text but it is reported in the legend of the figure. The chest was then closed in an air-tight way after inflation of the lung. Three days later, the animal was in good condition; it was placed on the table for taking a specimen of blood; suddenly it became cyanotic, very dyspnœic, and died in a very short time. In the roentgenogram (which is not given in his paper) "there were signs of atelectasis but without displacement of the heart or trachea." This absence of displacement was attributed by the authors to the "tearing of the flimsy and nonresistant mediastinum of the dog because of the rapid development of atelectasis." It is difficult to understand how the perfectly elastic mediastinum of a dog can be torn "by the rapid development of atelectasis"; it never happened in my experimental thoracic work; but even if such were the case, the heart and trachea should have been displaced just the same, as the mediastinum of the dog normally does not impede the passage of air or fluid through it. The diminution in size of the atelectatic lung is the only factor responsible for the displacement of the mediastinal contents, heart, and trachea in atelectasis. The only thing the mediastinal membrane itself can do is to resist this displacement; so that with a torn mediastinal membrane the displacement should be even greater. I personally believe that the animal died because of the sudden production of a pneumothorax due to the sloughing off of the pulmonary ligatures in the area from which the lung specimen was taken, and this would probably account for the non-displacement of the heart to the affected side. Besides, it would be very difficult to consider that in the denervated left lung the atelectasis was caused by vagus reflex transmitted "through the plexus of the right lung" while this latter lung remained completely sound.

Having cleared the way, I believe, of these 3 cases, I shall now briefly present what I consider physiological evidence against the so-called "vasomotor reflex."

Brodie and Dixon, in an exhaustive study of the innervation of the blood vessels of the lung, came to the conclusion that "pulmonary arterioles possess no vasomotor nerve supply." They have never obtained the least effect upon the pulmonary blood by exciting the white rami communicantes from the upper thoracic spinal nerves, the sympathetic chain between the successive ganglia, stellate ganglion, the annular loop of Vieussens, or the inferior cervical ganglion. Stimulation of the fibers at the root of the lung was ineffective. The results were the same in the dog, cat, or rabbit. Stimulation of the vagus was equally without effect, nor were they able to discover any vasodilator fibers to the lung in any of the nerves investigated. According to Starling, even if they exist, vasomotor nerves to the pulmonary vessels are of little importance.

It is not my intention here to discuss at length this phase of the subject. This question is taken up in detail in a forthcoming paper on the circulation in the atelectatic and pneumonic lung. Weber upholds the existence of vasomotor nerves to the lung, but Krogh has shown that all the "active vasomotor phenomena," supposed by Weber to occur in the pulmonary lobe enclosed in a plethysmograph with its bronchus tied, are due to increased or decreased output of the right heart and not to vasomotor nerves. Krogh concluded that "the evidence obtained from Weber's experiments is not favorable to the theory of pulmonary vasomotors." I personally believe that if the atelectasis were due to the mechanical expression of air caused by vessels so greatly dilated as to give the lung "an angiomatous" appearance (Bergamini and Shepard), then that lung should be at least as large and not smaller than the healthy one as it really is; in order to produce that mechanical expression of the alveolar air the dilated vessels would have to take the place of the actual alveolar space. Moreover, even if reflex vasodilation were possible in a healthy lung, inflation and not collapse of the lung would be produced. Vasodilation of the capillaries around the bronchi and narrowing of the bronchial diameter would produce emphysema for the reasons mentioned above; vasodilation of the alveolar

capillaries would not favor atelectasis, because vasodilation, by increasing the blood supply through the lung, would on the contrary tend to increase the air content of this organ. In a study of "vital capacity in intrathoracic therapy" Yates states that deliveries of blood through bronchial arteries and through the pulmonary arteries are controlled by the functional activities of the lungs which are proportionate to vital capacities. When the air cells are inflated, the capillaries are elongated and as they carry the air cells with them inflation is increased. These activities and reactions also take place in reverse order. In other words, blood flow and vital capacity follow parallel courses. Whatever may be the objection to this theory of air cell capillary mechanism which is supported by E. K. Dunham, Yates says that the fact remains that, in the normal lung, expansion is accompanied by an increased blood flow through the lungs.

This being the case, how can we reconcile the above facts with the microscopic findings in sections of atelectatic lung where there is to be found dilation of the small vessels? This fact was reported by several authors and verified by ourselves in our cases of experimental atelectasis in dogs (Figs. 1 and 2). We think that this contradiction is only apparent; *in fact the impairment of the circulation through the capillaries in atelectasis is progressive and proportional to the degree of absorption of air and of collapse of the alveoli.* The capillaries become more and more retracted as the atelectasis advances, and as a result there is stasis in the pulmonary arterioles. What appear to be dilated capillaries, in microscopic sections, are, in reality, dilated terminal arterioles; the circulation in the capillaries is actually greatly impaired, and the apparent vasodilation is not the cause but the result of pulmonary atelectasis. This was proved by Coryllos and Birnbaum (26) by injecting lipiodol or India ink into the jugular vein of the living animals in which atelectasis was produced. Lipiodol injected into the jugular vein penetrates the small arterioles but not the capillaries of the lung. Ten to forty cubic centimeters of lipiodol (iodized oil) or even more have been injected into the jugular vein, with survival of the animal from 3 to 10 minutes after the injection. Sufficient time was thus allowed the circulating blood to carry the lipiodol to the lung. Roentgenograms showed the finest details of the arterial tree (Figs. 3 and 4); but whereas the right heart was filled with lipiodol no trace was seen in the left heart, although the aorta was clamped for avoiding dilution of the lipiodol (Fig. 5), which shows that lipiodol does not pass through the capillaries. If now instead of using lipiodol the living animal is injected through the jugular vein with a 20 per cent solution of India ink in Ringer's solution according to the method of Krogh and Ehrich, this passes through the capillaries which are readily injected. Microscopic sections show that capillary circulation in the atelectatic lung is markedly impaired and that differences between the healthy and atelectatic lung are conspicuous (Figs. 6, 7, 8, 9, 10, and 11). As it is shown in these illustrations, the same procedure applied to the pneumonic lung yielded exactly the same pictures. This is a new and quite unexpected argument in favor of the conception developed by Coryllos and Birnbaum that lobar pneumonia should be considered as pneumococcic atelectasis (27). It throws a new light on the real mechanism of the impairment of the circulation in the consolidated pneumonic lung. From data to be published later with Dr. Birnbaum I can state here that this impairment would be due neither to thrombosis of the capillaries (Riebert, Kline and Winternitz) nor to the pressure exerted upon the capillaries by the exudate filling the air cells (Binger and Christie), but to retraction of the capillaries due to the collapse of the alveoli. This would explain the rapid re-establishment of the circulation when the lung is aerated again, both in pneumonia and atelectasis, and would offer a new argument in favor of the theory of the close relation between these two diseases.

A last argument against the reflex nervous theory lies in the fact that by rolling a patient back and forth in the treatment of atelectasis, as shown by Sante, a coughing spell is induced and in many instances with the expectoration of thick sputum a clearing up

of the lung rapidly ensues. Sante proposed this mode of treatment, which in several instances has proven successful. It is easy to understand how exudate flowing to dependent parts comes in contact with healthy bronchi and causes a spell of coughing which disrupts the obstructing column of mucus and transforms a complete obstruction into an incomplete one. This process in the mucous column is probably aided by a deep inspiration which follows a cough, because the forceful ingress of respiratory air into the bronchus could thus also create an airway by breaking up the column of mucus. Aeration of the affected lung would thus be initiated. But I do not see the mechanism by which the simple change of the position of the patient could abolish a bronchoconstrictor or a vasodilator reflex, nor can I understand how a reflex phenomenon would require so many hours to be established.

To the procedure advised by Sante can be compared the method used by Boulan and Cheret for prevention of atelectasis. They claim that by placing the patient in slight Trendelenburg position they avoided this complication. This was proved not only clinically but even by roentgenographic examination, the bases remaining clear and well aerated, whereas dark spots or massive opacity appeared in patients left in horizontal dorsal decubitus.

From the foregoing considerations it can be reasonably concluded that the so-called vasomotor reflex has no physiological or clinical foundation and cannot be considered as a cause of atelectasis.

COMMENT ON THE THEORY OF BRONCHIAL OBSTRUCTION

The fact that complete bronchial obstruction is followed by atelectasis has already received definite clinical and experimental confirmation, and for the reasons developed above I consider *bronchial obstruction as the only determining cause of the disease*. The difficulties that have stood in the way of determining the etiology and mechanism of atelectasis have been due, I believe, to the failure to distinguish between the determining cause and the contributing causes. The theory of bronchial obstruction applied both to postoperative atelectasis and pneumonia throws a new light on the etiology of both diseases and dispels the cloud hanging over their manner of production. This theory will be thoroughly discussed in dealing with the etiology of postoperative pneumonitis.

ETIOLOGY OF POSTOPERATIVE PNEUMONITIS

The term "pneumonitis" is purposely used here instead of "pneumonia" because in this group, besides well defined lobar pneumonias, we find a great number of transitory forms: bronchitides with areas of consolidation, bronchopneumonias with atypical evolution and symptoms, and pneumonias with confused symptomatology which may be difficult if not impossible to differentiate. They form a group of postoperative pulmonary complications which still puzzle the internists and surgeons. Their etiology and pathogenesis have been, and still are, subjects of intense research and discussion.

Their importance has been recognized only since the advent of the aseptic era. With the popularization of aseptic methods and general narcosis, it was recognized that in the absence of local infection the great majority of postoperative elevations of temperature were due to inflammatory conditions of the lung. At first it was believed that general narcosis, particularly with ether, was the cause of these pulmonary complications; Mikulicz, Poppert, and Czerny in Germany favored this view which was supported by experimental work of Poppert, who showed that ether could produce transudation and œdema in the lung and by the work of Snell who asserted that ether vapor diminishes or abolishes the bactericidal properties of the lung. Lichtenberg created the special term "Narcosepneumonie" for indicating this type of complication.

After the introduction of scopolamine-morphine anæsthesia and more particularly of local, regional, and spinal analgesia, surgeons were much alarmed on finding no reduction in the incidence or severity of postoperative pneumonitis. And although Neuber, Eiselsberg, Kummel, and others had the impression that with these anæsthesias

pneumonitis had greatly diminished or even disappeared, the papers of Finsterer, Hackenbruch, Reinhard, Roith, and others showed that its frequency and resulting mortality were about the same as with general anæsthesia. Lichtenberg (85), Demmer, Pfanner, and Gottstein believed that the incidence was even greater after local anæsthesia.

In the more recent papers of Mandl, Cutler and Morton, Cutler and Hunt, Whipple, Elwyn (46), Cleveland, etc., the facts mentioned are corroborated. The question, therefore, naturally arises as to the etiology and pathogenesis of postoperative pneumonitis.

The principal etiological factors brought forward are: Aspiration of septic contents of the mouth; hypostatic congestion; chilling; embolism and retention of mucous secretion in the bronchi.

ASPIRATION PNEUMONITIS

Despite the opinion of Chlumski, Hoelsher, and others, it is very improbable that a true aspiration pneumonia exists. I agree with L. Sante (1928) who says that aspiration alone could hardly explain the condition, since patients aspirating barium sulphate through bronchoœsophageal fistulæ never develop atelectasis. Likewise postoperative pneumonia is a rarity after tonsillectomy and there is abundant proof of the extreme degree of aspiration which frequently occurs during this operation. It is known that during general anæsthesia or even local anæsthesia of the larynx, much infectious material passes from the mouth into the trachea; Meyerson found blood in the bronchi of 75 of 100 cases bronchoscoped after tonsillectomy. Lipiodol passes into the trachea when instillated into the pharynx if the larynx is anæsthetized (Singer). Wessler has shown that aspiration produces suppuration and gangrene of the lung; but, as Elwyn (47) points out, aspiration does not explain the occurrence of pneumonia within a day or two of operation and especially of that type of pneumonitis which disappears after 3 to 7 days with no evidence of pus formation. Furthermore, pneumonia is more frequent after operations upon the abdomen than in operations on the mouth, head, or neck, performed either with general or local anæsthesia, although these procedures do reasonably favor aspiration. In discussing the embolic theory I shall quote respective figures of pneumonitis after operation upon the abdomen and other parts of the body, including the head and mouth, which will show clearly that aspiration of septic material, which to my mind constitutes the principal cause of abscess and gangrene of the lung, cannot be considered as the determining cause of postoperative pneumonitis.

PULMONARY HYPOSTASIS

With regard to *hypostatic congestion* (as in bedridden, cachectic patients or patients with advanced diseases of the cardiopulmonary circulation), it might be considered a contributing cause for development of pneumonitis because of the increased bronchorrhœa, posture, and impairment of the physiological defense of the lung. But to consider it as a determining etiological factor, *per se*, serves rather to confuse than to clarify the situation. Diminished respiratory activity, the recumbent position, or wasting illness alone cannot be the cause of postoperative pneumonitis which appears as well in strong young people with healthy cardiovascular systems. I shall only remark that under the same conditions atelectasis was said to develop and a similar discussion has been going on as we have already seen.

EMBOLIC THEORY

The embolic origin of postoperative pulmonary complications has had many staunch supporters. Lichtenberg (84), particularly, stressed its importance; Wolfer thought that the migration of thrombi from the pampiniform plexuses to the lung was the cause of postoperative pneumonias following the Bassini inguinal hernia procedure. Kelling considered that the great morbidity and mortality of postoperative pneumonia after abdominal operations for carcinoma of the stomach were due to the septic nature of the embolism in these cases. Rupp found at autopsy in 13,000 postoperative cases, 5 per cent having demonstrable emboli and infarctions in the lungs. Cutler and Morton in 1917

and Cutler and Hunt in 1920, in two thorough papers, tried to prove that almost all postoperative complications of the lung were due to embolism. The emboli, according to these authors, are formed in the operative field and from there are carried to the lungs "both by blood vessels and lymphatics." They explain the great number of pulmonary complications after operations upon the upper abdomen by the ease of formation of thrombi in that region because "laparotomy exposes surfaces incised in the outer world and evaporation and chilling take place easily." Furthermore, they believe that thrombi thus formed are easily mobilized and "set free because of the mobility of the structures of the epigastrium and the easy pathway to the lung and pleura from the upper abdomen both by the blood vessels and the lymphatics." They quote the experiments of Sabin demonstrating the facility with which lymphatic channels may carry sepsis or emboli from the epigastrium. According to these authors the small thrombi thus formed would be fixed in the lung, particularly in the congested or hypostatic areas of the organ.

Before any discussion of the embolic theory, it will be useful to review our knowledge on pulmonary embolism. Wharton and Pierson in their remarkable work on "Minor Forms of Pulmonary Embolism" state that the clinical aspect of embolism will depend upon four factors: the size of the embolus, the condition of the pulmonary circulation, the presence or absence of infection either in the lungs or the embolus, and the position of the artery which was occluded.

We are not interested here with the large emboli which cause immediate death—*la mort sans phrases* of the French authors—or with the medium size emboli which produce a typical infarction. The emboli which are meant by the supporters of this theory as etiological factors in the production of pneumonia or atelectasis are the *minor emboli.*

Let us see now in review the pathological physiology and pathology of the lesions produced in the lung by these "minor emboli." As Karsner and Ash have proved experimentally, the lesions which are produced in the lung by very small emboli vary with the condition of the circulation in the lung. They will produce significant lesions in the healthy lung "only when they lodge in vessels which are situated along the angular borders of the lobes." On the other hand, in the lung with impaired circulation and vascular "stasis, emboli of the same size produce *infarcts* no matter where they lodge, and after 24 hours they produce *the complete pathological picture of infarction* showing hæmorrhagic consolidation, pleurisy, and focal necrosis." In normal lungs, the lesions reach their maximum in 24 hours, never pass beyond the state of partial hæmorrhagic consolidation, do not develop focal necrosis or pleurisy, and resolution is prompt. Very often minor emboli pass through the lung without giving rise to any lesions or symptoms, exactly as happens in experimental embolism in dogs, in which even large sized aseptic thrombi introduced into the jugular generally produce no lesion. It is known how difficult it is to produce embolic lesions of the lung in these animals with small or even good sized aseptic and often even with septic blood clots.

In the congested lung, in the human, minor emboli according to their nature may produce aseptic or septic lesions. In the former, the pathology will be exactly the same as infarctions; in the latter, suppuration with septic necrosis may follow. This pathology is very different from that of postoperative pneumonia. Whipple has given an excellent description of the latter and rightly compared it to the mild form of medical pneumonia, known as *maladie de Woillez*, so well described by Carriere. Maurice Letulle gives a very good résumé and photographs of it in his excellent book, *Pathologie du Poumon.* Furthermore, the clinical symptomatology of embolism is characteristic; Wharton and Pierson state that the onset clearly distinguishes this form of pulmonary embolism (minor) from the postoperative inflammatory lesions and it is during the first days of the attack that the diagnosis should be made. Infarction occurs late in convalescence, after an uneventful week or two, seldom as early as the third or fourth day. The attack occurs suddenly with pleuritic pain below the scapula, tachypnœa, and slight elevation of

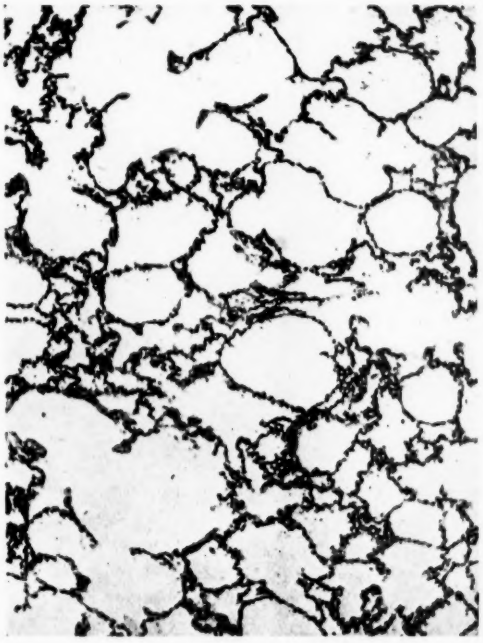

Fig. 6. Dog 82. Photomicrograph of a section of lung injected with India ink—Ringer solution. Section is not stained. The capillaries are perfectly injected. Notice the size of the alveoli outlined by the capillaries. Section taken from the healthy non-atelectatic lung.

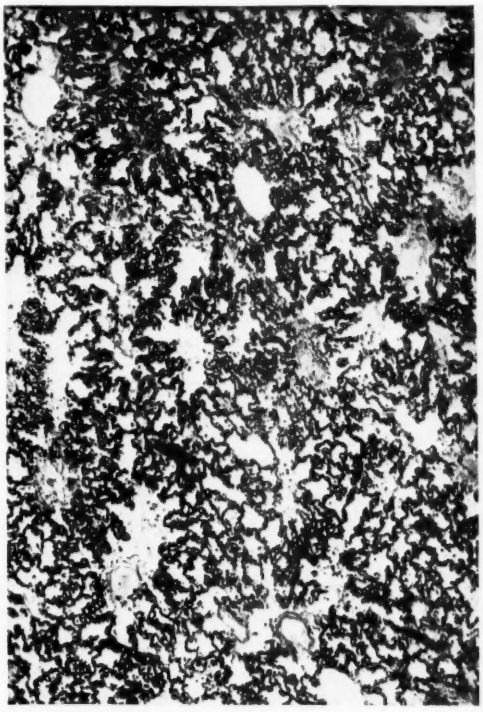

Fig. 7. Dog 82. Atelectasis. India ink, Ringer solution, injected into the jugular vein. Section of a portion of the atelectatic lung not completely apneumatic. Notice the collapse of the alveoli; shrunken capillaries but still permeable. Dilated vessels (precapillary arterioles) make their appearance.

temperature, without any physical signs until the second or third day after the onset. At this time there develops the characteristic friction rub and non-productive cough or with sputum more or less tinged with bright or dark red blood. Following this, after the second or third day, the condition gradually improves. In the great majority of cases the physical signs are friction rub, which is the most reliable symptom, present in about 50 per cent of these cases, and often impairment of the percussion note. In postoperative pneumonitis, on the other hand, the onset is within 12 to 24 hours after operation, is accompanied by cough, dyspnœa, often cyanosis, as a rule a rise of temperature, and absence of friction rub; furthermore, the physical signs of consolidation are never absent. Very often the patient gives a history of "previous cold" before operation. The differences between this syndrome and infarction are so marked and so characteristic that a careful examination of the patient must establish the diagnosis. Cutler and Hunt (31) make this distinction themselves by separating the cases of infarction from other postoperative complications, although I believe that their two cases of postoperative pleurisy seem to be clear-cut cases of "minor embolism." The cases of these authors described as pneumonia, bronchopneumonia, and bronchitis are, I believe, all cases of postoperative bronchitis with atelectatic consolidation. Cutler and Hunt (31) themselves admit this point by stating that the "dividing line between pneumonia, bronchopneumonia, and bronchitis is not always clear"; out of 55 cases of postoperative pulmonary complications of these authors, 42 are called bronchitis, bronchopneumonia, and pneumonia. No one

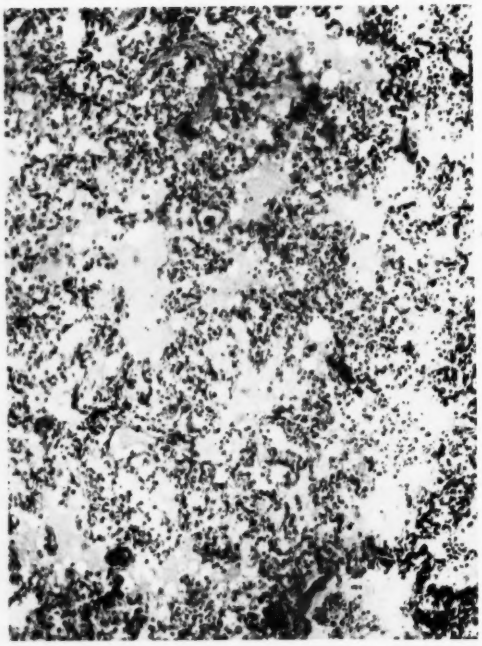

Fig. 8. Dog 82. Atelectasis. India ink, Ringer solution, injection through the jugular vein. Specimen of completely apneumatic portion of the lung. Notice that capillary circulation is greatly impaired, but not completely suppressed. Dilated vessels (precapillary arterioles) are still more visible than in Figure 7. (Figures 6, 7, and 8 are taken under some enlargement.)

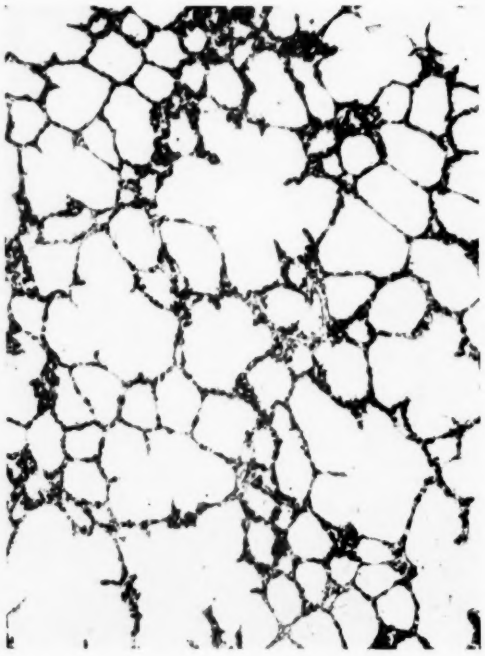

Fig. 9. Dog 83. Experimental pneumonia. India ink, Ringer, solution injection in jugular vein. Section from healthy non-consolidated lung. The capillaries well injected outline the patent alveoli. Notice their size for comparison with Figures 10 and 11.

of these cases, for the reasons given, seems to be secondary to emboli; a careful study of the histories of these cases can leave no doubt about this point. I feel convinced that this fact would have been more apparent had the authors recorded their radiographic findings and the bacteriological examinations of the sputum, particularly for pneumococcus. In order to illustrate this statement I give here the histories of two of their cases. The first is diagnosed as pneumonia, the second bronchopneumonia.

L., a male, 28 years of age, with left nephroptosis; lungs negative before operation. Operation for partial decapsulation and nephropexy were done under ether-oxygen anæsthesia. Recovery was good. Twenty-four hours later the temperature was 103.6 degrees, pulse 143, respiration 40. Signs of consolidation were present at the right base. The sputum was negative for tubercle bacilli; white blood cells on tenth day were 12,000. Recovery was by lysis on the eleventh day.

In female, 50 years of age, the lungs were normal, but the heart showed a soft systolic murmur. Under ether anæsthesia, an operation was performed for an ovarian cyst. On the first day after operation there were dullness and bronchial breathing at the right base. The temperature was 101 degrees, pulse 100, respiration 38. Two days later consolidation of the entire back was present. There were râles but no dullness on the left side. The sputum was negative for tubercle bacilli but a great number of diplococci were found. She was discharged against advice.

In both cases the onset was within 24 hours of an aseptic operation. The right base was affected in both; in the second the whole right lung 48 hours after the onset. A roentgenogram in this latter case would probably have revealed the signs of atelectasis. Many diplococci were found in the second case. I do not see how these cases can fit into the symptomatology or pathology of embolism.

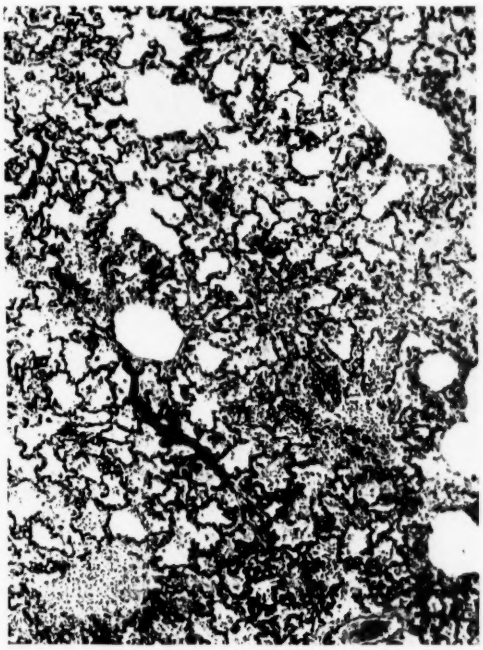

Fig. 10. Dog 83. Experimental pneumonia. Section of portion of the lung not completely consolidated. Alveoli and capillaries shrunken, capillaries only slightly impaired. Notice the presence of dilated vessels (precapillary arterioles.) Compare with Figure 7.

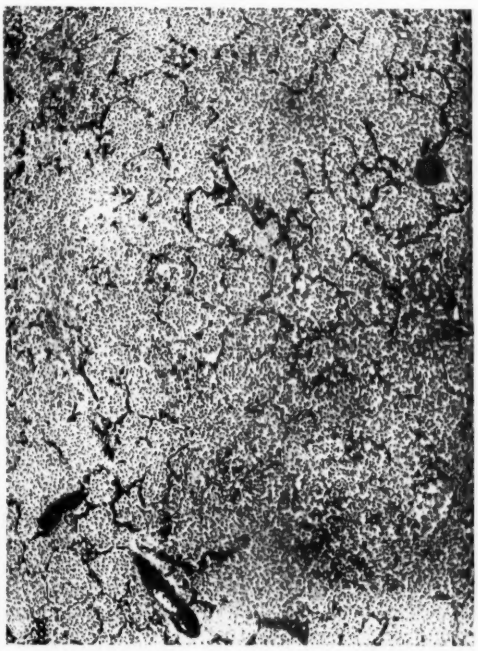

Fig. 11. Dog 83. Experimental pneumonia. Complete (gray) consolidation. Circulation greatly impaired but not completely stopped. Notice the great number of dilated vessels. Compare with photomicrograph of Dog 82 shown in Figure 8.

If we analyze the case histories of postoperative pneumonia, bronchopneumonia, atelectasis, and even more or less complicated bronchitis, which all form a well defined group designated by Wharton and Pierson as "inflammatory pulmonary complications," we must conclude that they are certainly "bronchogenous" and not of embolic origin. There are further arguments in favor of this opinion: First, why are these lesions exceptional after operation upon the extremities, the head, and even the mouth if they are of embolic origin, and so frequent after operations upon the abdomen, more particularly the upper abdomen? In 97 cases of pneumonia reported by Whipple, 88 occurred after laparotomies. Cutler and his collaborators report that the incidence of pneumonia was 1.12 per cent after operations upon different parts of the body other than the abdomen, 4.48 per cent after low abdominal operations, and 7 per cent and 8 per cent after operations upon the upper abdomen. Mandl's statistics on 1,300 cases give 2.7 per cent as the incidence of pneumonia after operations upon the extremities, the head, the mouth, and the neck, and 10.5 per cent after upper abdominal operations under general anæsthesia. Elwyn (46) reports on 0.7 per cent incidence after operation upon the extremities, head, mouth, and neck, 6.29 per cent incidence after abdominal operations, and 13.8 per cent after gastric operations. Head and Powers presented a clear explanation of these clinical facts by showing the variation of vital capacity after operation. They found that the greatest changes were produced in abdominal operations and the reduction was proportionate to the extent of the procedure and the proximity of the operative field to the diaphragm. The majority of authors who deal with post-

Fig. 12. Dog B 47. Twenty-four hours after blocking the right bronchus. Complete right atelectasis. Occluding balloon filled up with sodium bromide solution is clearly visible in right bronchus.

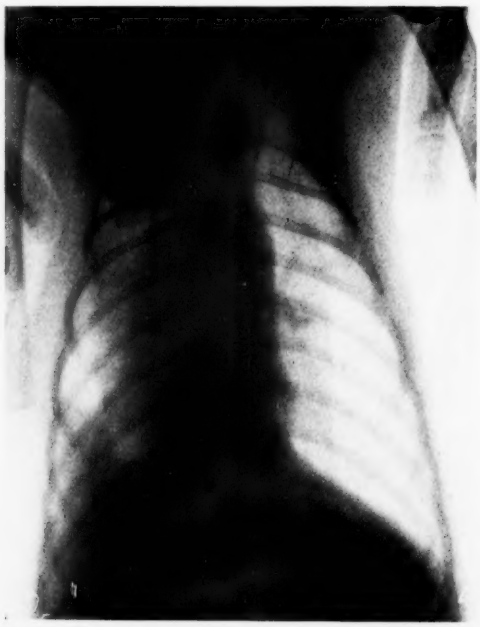

Fig. 13. Dog B 5. Right lobar pneumonia 24 hours after insufflation of 10 cubic centimeters of the pneumococcus type 1 culture into the right bronchus. Notice the displacement of the heart to the right and the marked elevation of the diaphragm on the right. The same results were obtained when 1 cubic centimeter of concentrated culture was used.

operative pneumonitis or atelectasis are not in favor of the embolic theory for either of them. Henle, in 52 autopsies in a series of 143 surgical pneumonias, found only 5 cases of infarction. A. C. Whipple, in 7 autopsies of 25 fatal cases of postoperative pneumonitis, found no embolism. Furthermore, in cases in which pneumonia or atelectasis and embolism existed together it was possible clearly to distinguish one lesion from the other. Churchill reports a case of embolism complicated by atelectasis, in which the embolism (sharp pain, rise of temperature, loud crunching friction rub at the right base, blood streaked sputum) was followed after 4 days by signs of atelectasis. This case shows, according to Churchill, that "even when atelectasis accidentally follows embolism, the two conditions are distinct and can be readily recognized." Conversely, embolism may complicate atelectasis as in Case 5, of Jackson and Lee. A colored man with primary carcinoma of the œsophagus and extensive metastasis in the liver died suddenly after a sharp rise of temperature. At autopsy the findings were: "a fresh wedge-shaped hæmorrhagic infarct in the left lung and complete atelectasis of the right lower lobe." It might be objected that the above facts concern atelectasis and not pneumonia. I shall answer this objection in the next part of this paper, where atelectasis and pneumonitis will be compared. However, in closing the subject of possible embolic origin of atelectasis, I quote Churchill who aptly remarked: "We see no mechanism whereby an embolus can produce pulmonary collapse." Karshner and Ash have not been able to produce immediate deflation of the lung in minor embolism of this organ. Schlaepfer, by ligating a branch of the pul-

Fig. 14. Dog 251. Complete left atelectasis 24 hours after blocking left bronchus with balloon. Notice clear triangular area *T*, due to encroachment of normal lung upon affected side and presenting a curious similarity to the sonorous triangle described by Lord in pneumonia.

Fig. 15. Dog B 27. Left lobar pneumonia 22 hours after insufflation of 1 cubic centimeter of concentrated culture into left bronchus. Notice the clear area at the left base described under Figure 2.

monary artery, succeeded in producing a gradual shrinkage of the affected portion of the lung with a noticeable displacement of the diaphragm only a week after and without infarction. Elwyn (46) also discards embolism as a factor in the production either of atelectasis or of postoperative pneumonia. He says that occasionally he has observed cases of pulmonary infarction following operation, "but they were definitely diagnosed as such." Among his 89 cases of postoperative pneumonia none could definitely be said to have been caused by embolism from the operative field. Wharton and Pierson protest against the assertion made by authors who consider that the cause of any or almost any postoperative pneumonia is due to "showers of minor emboli" and insist upon the fact that "it is as a rule possible to establish a diagnosis between embolic lesions and inflammatory pneumonia."

It is not necessary to give further quotations and arguments against the embolic theory. I firmly believe that minor embolism as an etiological factor in postoperative pneumonitis cannot be admitted. It was necessary to insist upon this point in detail because, in my opinion, the embolic theory should be definitely discarded in order to clear the way for a better understanding of the etiology, nature, and treatment of postoperative atelectasis and pneumonitis. In this way only can we avoid unwarranted conclusions concerning the treatment of pneumonitis and *atelectasis*.

It is not deemed necessary to discuss the opinions that *chilling* or *irritation of the bronchial mucosa by anæsthetics* can by them-

Fig. 16. Case 73,814, New York Hospital. Postoperative massive atelectasis, right side. Note dense opaque shadow which completely obscures right chest. Note marked displacement of trachea to right and pulling over of heart shadow to right.

selves produce postoperative pneumonitis. As to the theories of *passive or active congestion* as principal causes, the same question arises, namely, what is the mechanism of production of postoperative pneumonitis in these conditions? A study of the similarities between atelectasis and pneumonia will help us, I believe, to give a satisfactory solution of the problem of their production.

SIMILARITIES BETWEEN POSTOPERATIVE BRONCHITIS, ATELECTASIS, AND PNEUMONITIS

The kinship between these complications—bronchitis, atelectasis, and pneumonitis—more or less apparent when we study them clinically, becomes striking when we investigate them experimentally. As Coryllos and Birnbaum have shown (27), it is impossible in a roentgenogram of a dog's chest to make a differential diagnosis between experimental pneumococcic lobar pneumonia and massive atelectasis. Figures 12, 13, 14, and 15 clearly prove this fact.

There are so many similarities between postoperative bronchitis, atelectasis, and pneumonitis that they cannot and must not be considered as coincidences; they require a more careful analysis and investigation than has formerly been given them.

The similarities are *clinical*, *pathological*, and *etiological*.

CLINICAL

Atelectasis and pneumonitis appear within 24 to 48 hours after operations, performed particularly upon the abdomen. In the literature, the statistics on postoperative atelectasis of W. Pasteur, Armstrong, Scrimger, Churchill, Scott (124, 125), Holmes, Mastics et al., (90) Jackson (69), Lee, Leopold (81, 82), Elwyn, Hirschbrock, Rigler, Mason, H. Santee, Bergamini and Shepard, Ball, Roland and Cheret, etc., clearly show a great predominance of abdominal operations as forerunners of the disease. In 134 cases of atelectasis compiled from the literature, the following incidence was found:

	Cases
Appendectomies—septic or aseptic	42
Hernias, simple or strangulated, inguinal or ventral	36
Gastric or duodenal procedures and exploratory-laparotomies	6
Cholecystectomies	17
Hysterectomies or salpingectomies and cæsarean sections	17
Thyroidectomies	5
Kidney operations	3
Rectal operations	2
Perineal operation	1
Injury of hip	1
Fracture of cervical spine	1
Fracture of pelvis	1
Tumor of thigh	1
Axillary abscess with streptococcus bacteræmia	1
Total	134

It should be borne in mind that these statistics are incomplete, since the diagnosis of atelectasis is still relatively infrequently made in the United States and even less frequently at Continental medical centers. It is interesting in this regard to quote Pasteur (101), who, in reporting his statistics from the surgical service in Middlesex Hospital, says, "Mr. Simmonds (the surgeon) considers, from the description given in the notes, that 20 cases regarded as pneumonia were probably examples of massive collapse."

The onset. Within 24 to 48 hours after operation, pain and dyspnœa, a variable degree of cyanosis, and elevation of temperature may appear. The above signs and symptoms are

Fig. 17. Right pneumonia in human (lower and middle lobes). Notice the displacement of trachea and heart to right and the elevation of the right diaphragm. Compare with Figure 16.

Fig. 18. Case 100,390, New York Hospital. Postoperative massive atelectasis, left side.

common in both. It is difficult in their initial periods to distinguish atelectasis from pneumonia, or even from bronchitis. As proof, I quote the description of postoperative bronchitis given by Cutler and Hunt (32, p. 23): "The onset of bronchitis is accompanied by cyanosis, excessive perspiration, the chest is often full of moist râles. It starts always within 48 hours after operation and subsides usually by lysis within 3 days to 2 weeks." Cough is not a constant symptom in atelectasis and pneumonia. In a footnote on F. G. Blake's article on pneumonia, in which it is said, "Cough is not frequent in the earlier stages of pneumonia," Blumer remarks: "Turning the patient on the side with the affected side upward so frequently causes cough that I regard it as a diagnostic sign of some importance" (page 9). Is it not common knowledge that exactly the same thing happens in atelectasis? On this latter fact L. Sante based the treatment of rolling the patient back and forth.

Evolution. This is very similar in both diseases and lasts from 2 to 13 days, ending

Fig. 19. Case 90,744, New York Hospital. Left lower lobe "lobar pneumonia," from the Medical Ward. Notice displacement of the heart and trachea to the left. Compare with Figure 18.

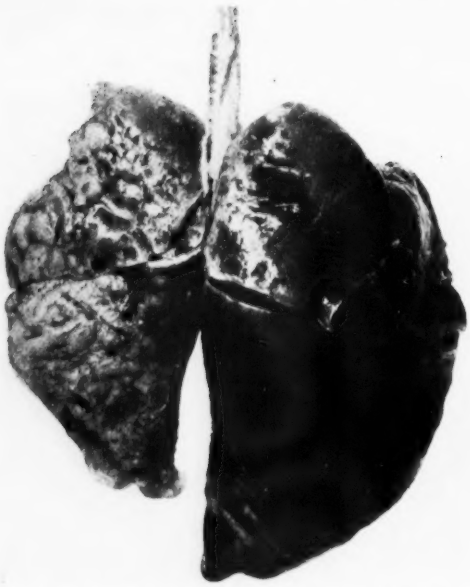

Fig. 20. Right lobar pneumonia in monkey's lung. Posterior view. (After Blake and Cecil.)

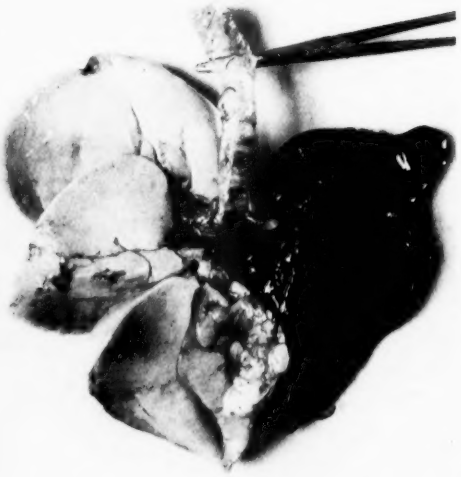

Fig. 21. Experimental pneumococcic pneumonia in dog, left lung consolidated. Lung extracted, after clamping the trachea (before the opening of the chest). Notice the difference in the size between the consolidated, airless, dark colored left lung and the light colored, normal right lung.

by crisis or lysis. But there are even more striking similarities. The lobar localization in both atelectasis and pneumonia was the one which impressed the writer the most at the beginning of his experimental investigation on atelectasis. Why does this lobar distribution occur in atelectasis and in postoperative pneumonia as well as in medical lobar pneumonia? Little thought about this point and still less explanation of it have so far been given. We shall return to this question later.

The physical signs by percussion and auscultation are no less impressive by their similarities. At the beginning of a pneumonia there are: dullness, with slight tympanitic resonance, and absence of breath sounds. In lobar atelectasis we have these signs throughout the disease. Likewise, fine râles, bronchial breathing, and increased fremitus are apt to develop or be absent both in postoperative pneumonia and postoperative atelectasis. J. R. Bradford (13)[1] divides the physical signs of atelectasis into three periods: (1) signs of retraction and immobility of the affected side, weakness or absence of breath sounds, and displacement of the heart often extreme; (2) weakness of the breath sounds has been replaced by loud tubular or amphoric breathing together with increased vocal fremitus, loud bronchophony, pectoriloquy, and transmitted spoken voice; (3) stage when the lung is expanding; abundant râles may be present over the area where tubular breathing is marked.

The same author considers atelectasis as "a possibility in pneumonia" and reports the autopsy of a case in which there was "a pneumonia in the upper left lobe and atelectasis in the lower"; and further he states that "pneumonia may complicate atelectasis and then is limited to the affected lobe." Norris and Landis speak of massive atelectasis as a complication of pneumonia, and Reynolds (1871) describing consolidation in newborn children insists on the point that the distribution of the affected lobules is in direct relation to the condition of the corresponding bronchial tubes. It is often impossible from the case histories to differentiate atelectasis from pneumonia. For illustration of the above statements, I give the history of a case called "atelectasis" (Case 1, Churchill, 21):

[1] In W. E. Lee. Ann. Surg., 1924, lxxix, 524.

V. S. A woman, 20 years old, gave no history of past or present respiratory infection, and a preoperative roentgenogram of her chest was negative. A left oophorectomy and right salpingo-oophorectomy with routine appendectomy were performed for bilateral, multilocular adenocystomata. Ether anæsthesia with gas induction was employed. During the operation the respirations were rapid, with excessive mucus. On the evening of the following day, there was a sudden rise of temperature, pulse, and respiration, accompanied by dyspnœa. Examination showed a limited excursion of the right chest with flatness to percussion and diminished breath sounds below the clavicle. The heart was displaced to the right. The next day the patient was improved and she raised a small amount of purulent sputum containing *pneumococci* and streptococci. Sonorous râles were present throughout the lungs and below the angle of the scapula. On the right there were bronchial breathing, ægophony, increased voice sounds, and normal tactile fremitus. A roentgenogram taken 2 days after the operation showed increased density of the right chest, especially at the base, and displacement of the heart and mediastinum.

Is not this case, diagnosed "atelectasis," strikingly similar to an atypical case of pneumonia of the right base? Auscultatory symptoms of consolidation and pneumococci in the sputum were present. In other cases of the same author, pneumococci types 3 and 4 were found in pure culture. The sign on which the differential diagnosis between atelectasis and pneumonia was based was displacement of the heart and trachea to the affected side. But it has already been shown that this sign may as well be present in pneumonia, particularly in children, and it is always present in experimental pneumonia in the dog (Figs. 13, 15). At the onset of atelectasis, says L. Sante (119), the condition may resemble lobar pneumonia; and, "if alveolar absorption takes place rapidly and there is still some obstruction of the bronchi by secretion, displacement of the trachea may be present in pneumonia as a manifestation of atelectasis; under these circumstances *lobar pneumonia* in the resolving stage *cannot be differentiated from massive collapse.*" Conversely, when the upper lobe alone is atelectatic "displacement of the heart may be negligible and deviation of the trachea the only symptom." Pratt and Bushnell say (107, p. 139): "While the pneumonic process is still localized, the loss of expansibility of the affected lobe tends to produce a displacement of the heart toward the lesion. Later in the disease when the hepatized lobe has increased in volume, it tends to push the heart away." However, they add, "such displacement is usually not well marked."

E. Jaches found in roentgenographic examinations that "massive atelectasis caused practically the same density of a lobe or the entire side of the chest as that seen in lobar pneumonia," but, he adds, "it may be distinguished from the latter by the displacement of the heart to the affected side and elevation of the diaphragm." L. Sante expresses a similar opinion. S. Leopold (82), discussing the differential diagnosis between pneumonia and atelectasis, says, "This is the condition with which massive pulmonary collapse is most frequently confused, as evidenced by the experience of numerous observers who, after having their attention attracted to massive collapse, have gone back over their records of 'postoperative pneumonia' and discovered that in some cases the symptoms, the physical findings, and roentgen-ray studies, were typical of massive collapse. The symptoms, pulmonary physical findings, and leucocytosis may be regarded for clinical purposes as so similar that on *these findings a differential diagnosis is impossible. The displacement of the heart and mediastinal structure towards the affected side in case of massive collapse is the diagnostic point of differentiation.* The massive degree of involvement in cases of collapse, the absence of the toxicity one would expect in lobar pneumonia, particularly in the postoperative type, are of some importance when these cases are considered as a group, *but would not be very helpful in an individual case.*"

It would seem from the above that the only diagnostic physical sign between these two postoperative complications, atelectasis and pneumonia, is, on last analysis, the displacement of the heart and the trachea to, and elevation of the diaphragm on, the affected side in atelectasis. But even this sign is not at all pathognomonic. In several instances, "inexplicable displacement of the mediastinum and its contents to the affected side and elevation of the diaphragm" have

been found in pneumonia. Thoenes, in 1922, reported 11 cases of lobar pneumonia in which roentgen-ray examination showed displacement of the heart to the affected side, more particularly in early life. St. Engel pointed out that elevation of the diaphragm to the affected side in lobar pneumonia is of common occurrence. Wallgren insisted upon displacement of the heart toward the affected side in unilateral croupous pneumonia in children. Griffith (54) gives the history of a child less than a month old in which the diagnosis between atelectasis and pneumonia was impossible, and he concludes that lobar pneumonia *may at times be capable of producing similar, if not perhaps as marked, roentgen-ray appearances as seen in massive atelectasis*. The same author, in a more recent paper (1927), reports 40 cases of pneumonia in young children. Sixteen among them presented displacement of the heart to the affected side. All the authors mentioned explained the displacement by compensatory hyperdistention of the healthy lung with the exception of St. Engel, who is the only one to suggest that "it might be dependent upon a diminution of size of the affected lung caused by a reflex interference with respiration on the side."

Among roentgen-ray plates of pneumonia at the New York Hospital, obtained through the kindness of Dr. W. W. Belden,[1] were found a great number showing displacement of the heart and trachea to the affected side, and in almost all, elevation of the diaphragm was found (Figs. 16, 17, 18, 19). In a series of slides of Dr. T. C. Roper[1] (prepared in 1915) dealing with evolution of lobar pneumonia in children, not only did I find displacement of the heart and elevation of the diaphragm to the affected side in practically every one of these, but, furthermore, there was a characteristic return of the structures to their normal sites as the diseased lung healed. The roentgenograms given by O. T. Pickhardt in his paper on "Unresolved Pneumonia" are also of interest in this respect. One can clearly see in this author's illustrations displacement of the heart to the affected side and homolateral elevation of the diaphragm (Figs. 2, 4, 6, and 8) with the return of these structures to normal position after healing of the lesion (Figs. 3, 5, 6, 13 in the paper of this author).

[1] Personal communication.

There is no doubt that displacement is not due to a *primary* compensatory emphysema or overdistention of the healthy lung, compressing the consolidated lung. A hyperdistention of the healthy lung certainly exists, but this is secondary and due to the *decrease in size of the affected lung*. The view that the pneumonic lung is decreased in size, contrary to what is generally believed and taught in textbooks (Fig. 20), is supported by the fact that in uncomplicated lobar pneumonia there is never displacement of the heart toward the healthy side, nor flattening of the diaphragm, findings which one should expect if the diseased lung were really enlarged. The idea that the pneumonic lung is larger has been perpetuated from one textbook to another, and from generation to generation, because the lungs are examined on the autopsy table after their extraction from the thoracic cavity and without the trachea having been previously clamped. It is natural that under such conditions, with the negative intrapleural pressure eliminated, the healthy lung collapses readily whereas the consolidated lung cannot. It is easy to verify this fact by removing the lungs of a person shortly after death from pneumonia, after having previously completely occluded the trachea with clamps. One should carefully avoid undue manipulation of the healthy lung and proceed with dispatch, because the air diffuses through the surface of a healthy lung very rapidly, as Lichtheim has shown. Figure 27 gives the relative sizes of consolidated and healthy lungs in experimental pneumonia in the dog.

Thus the last and only remaining differential or "pathognomonic" sign between atelectasis and pneumonia would seem to be neither pathognomonic nor characteristic of atelectasis as is commonly supposed.

PATHOLOGICAL

In the pathological examination we find marked similarities in pneumonia and atelectasis. In both conditions the lung is airless, apneumatic, consolidated, fleshy, friable, and

TABLE I.—STATISTICS OF MASTICS AND OTHERS

Lobe	Number	Per cent
Right lower	25	50
Left lower	10	20
Right upper	2	4
Left upper	3	6
Right middle and lower	7	14
Right upper and lower	2	4
Right upper and both lowers	1	2

TABLE II.—STATISTICS OF ELWYN

Lobe	Number	Per cent
Right lower	35	55.5
Left lower	17	28
Both lowers	7	11
Right upper	1	1.5
Left upper and left lower	1	1.5
Right upper and both lowers	2	3.5

sinks in water. In both, pneumococcus, particularly group 4, is almost constantly present, and it is curious that this fact has not attracted more attention. Whipple has shown that in postoperative pneumonia "sputum, as a rule, is a yellow mucus and usually shows pneumococcus, group 4, in both the pre-operative and postoperative specimens." Cleveland expresses the same opinion. In atelectasis the sputum presents the same characteristics.

Churchill reports 9 cases of atelectasis and in practically every one in which an examination of the sputum was made, pneumococcus, group 4, was found. In one case pneumococcus, type 3, was present—an unusual finding in postoperative pneumonia.

In the case of Hearn and Clerf, which was bronchoscoped seven times within 36 days, the secretion contained gram positive diplococci. In the cases of Lee, Tucker, and Clerf, the mucous secretion obtained by bronchial aspiration contained pneumococci, and when injected into the bronchus of an anæsthetized dog, atelectasis was produced. On the other hand, in cases of medical pneumonia in the human, bronchoscoped in the services of Drs. A. Lambert and L. Conner by Dr. J. D. Kernan and myself, we constantly found the bronchus corresponding to the pneumonic area occluded with thick exudate as in atelectasis. After bronchoscopic aspiration of a few centimeters of exudate, often a great amount was expectorated by the patients exactly as in cases of atelectasis (24).

Another argument in favor of the identity of these two conditions is the similarity of localization. Table I shows the statistics of Mastics, and others, in 50 cases of atelectasis.

In Scott's statistics of atelectasis (40 cases) only the side is mentioned: the right side was involved in 31 cases, or 78 per cent; the left side was involved in 9 cases, or 22 per cent.

Table II shows the statistics on postoperative pneumonia of Elwyn (63 cases, following operations performed under local and general anæsthesia).

The comparative study of Tables I and II shows clearly the striking similarities between these two conditions, so far as their localization is concerned.

ETIOLOGICAL

In order to avoid unnecessary repetition we will merely show that in the great majority of cases the two conditions cannot be differentiated from the etiological standpoint. A number of cases taken from different authors will illustrate this point. The following are two cases of Rigler.

CASE 4. Twenty-four hours after a bilateral salpingectomy, the temperature rose to 100 degrees. The next day it was 101 degrees, and patient had a cough, was cyanotic, and showed signs of consolidation of the right lung. Roentgenograms showed the right lung opaque, mediastinum markedly displaced to the right. On the third day, the temperature rose to 104.2 degrees, with 15,000 white cells. The crisis occurred 10 days after the onset, and 2 days later the right lung was clear and the heart in normal position. Two days later symptoms of empyema developed, displacing the heart to the left side.

CASE 5. Typical right massive atelectasis, 24 hours after salpingectomy, until third day, when rusty sputum appeared with distinct signs of consolidation of the right upper lobe. Roentgenograms showed upper lobe pneumonia with haziness of the medium and lower right lobes. There was a marked displacement of the mediastinum to the right and a very high right diaphragm.

Rigler thinks that in these cases pneumonia complicated atelectasis; that the first was a case of typical atelectasis, but because of the empyema which followed 2 days after crisis, Rigler presumes that the elevation of temperature from 101 to 104.2 degrees on the third day must have been due to the development of pneumonia as a complication. I

cannot agree with Rigler because there are a great number of cases of atelectasis with a temperature of 104 degrees, and more with a 15,000 white cell count. Shall we consider these cases as atelectasis or as pneumonia? We know that in almost every case of atelectasis, pneumococcus is present in the bronchial exudate, and often in pure culture. The fact that the development of pneumococcic empyema is unusual in atelectasis seems surprising, and this is due, I believe, to the low virulence of the infecting agent and to the rapid liberation of the bronchi. In Case 5 of Rigler we have the same phenomenon as in the cases of Elwyn (46) given later. In this patient we have a clear cut case of postoperative atelectasis, which, because of the presence of virulent pneumococci in the exudate occluding the bronchus, was transformed into lobar pneumonia. N. B. Gwyn, in 1926, reported cases of atelectasis complicating pneumonia.

CASE 1. A woman, 60 years old, with lobar pneumonia, had complete consolidation of the right lobe. About the fifth day of the disease she was suddenly seized with dyspnœa, prostration, and cyanosis, and the upper lobe, which was formerly overexpanded, was now apparently solid. The physical signs were those of a newly consolidated area, but the heart was displaced to the right, beating to the right border of the sternum. Clearing began in 6 hours and the heart returned to its normal position in 4 days.

CASE 2. Patient entered with signs of a small area of consolidation at the left base. During the night there occurred an attack of dyspnœa and in the morning the involvement seemed more extensive. There was a marked displacement of the heart; the apex was felt in the left axillary line, the left chest was flatter than the right. Roentgenograms showed a typical picture of atelectasis. Healing occurred after a week, with return of the mediastinum to its normal position.

CASE 3. A boy of 6 had pneumonia in the left lower lobe. Involvement of the left apex occurred at the end of 2 weeks, without added distress. The chest was completely solid, sounds were tubular but varying. From day to day the presence or absence of râles was noted. The left chest was sunken and motionless. The right chest was overdistended and clear. The heart was displaced to the left and upward. The left diaphragm was elevated to the level of the third rib in front. The left lung was very solid behind; puncture gave no fluid. Three days later examination showed that resonance was appearing in the midback and that the heart was lower in the chest. Roentgen-ray picture revealed the heart in normal position, diaphragm lower.

In these cases atelectasis complicated an already existing pneumonia. How would it be possible to explain the pathogenesis of atelectasis in these cases by vasomotor or by reflex nervous obstruction of the bronchioli of the affected lobe or by embolism? I believe that in these cases a temporary obstruction of another bronchus occurred which produced an atelectasis of the lobe corresponding to that bronchus. If, in these cases, the bronchial obstruction had been of longer duration and the pneumococcus virulent enough to produce pulmonary cellulitis instead of simple apneumatosis, we would have had an ordinary extension of the pneumonic process to the newly affected lobe. Here, on the contrary, has happened exactly what occurs in postoperative atelectasis, where, although pneumococcus is present, lobar pneumonia does not develop, because of the rapid liberation of the bronchus from the mucous "plug." This same mechanism can easily explain the production of the so-called abortive forms of pneumonia. This opinion seems corroborated by the fact that pneumonia can complicate atelectasis as is shown in the following cases of Elwyn (46).

CASE 1. The day after a left herniotomy the patient developed a cough and had pain in the right chest. Breath sounds were absent anteriorly and over the right chest and below the third rib. There was marked dullness and diminished breath sounds below the angle of the scapula posteriorly. The heart was displaced to the right. These symptoms persisted for 4 days, when dullness, bronchial breathing, and râles over the lower half of the right lower lobe appeared. The roentgenograms showed a dense shadow over the lower half of the right lung and the heart and the trachea and mediastinum displaced to the right.

CASE 2. Colostomy was done for carcinoma of the ileocæcal junction on June 20. A roentgenogram taken previous to the operation showed the chest to be normal. The same day the temperature rose to 102 degrees. The day following the operation, a physical examination showed breath sounds markedly diminished over the lower two-thirds of the right lung anteriorly and posteriorly. On percussion there was a tympanitic note in front and dullness from the spine of the scapula to the base posteriorly. Roentgenograms on the third day showed heart and trachea displaced to the right and a very high diaphragm on the same side. On

the ninth day after operation bronchial breathing was heard over the lower half of the right lower lobe. From this day on the breath sounds gradually became more distinct over the remainder of the lung, and the bronchial breathing over the right base gradually diminished. The heart came back to its normal position after some time.

Elwyn (46) considers the above cases as of "uncommon occurrence." I do not agree with this opinion and consider these cases much more frequent than generally believed. If they are not reported more often it is because special attention had not been given to them. In these two cases of postoperative massive atelectasis, the middle and lower lobes were involved because of the occlusion of the right bronchus below the level of the upper bronchus; then because of the dislodgement of the "plug," the middle and lower lobes became aerated, with the exception of that portion of the latter dependent upon the first posterior bronchus, which remained occluded. It is of no little importance that the persistence of the occlusion was due to increased virulence of the pneumococci; it is known that viscosity of the exudate and amount of fibrin depend upon the degree of virulence of the pneumococci. The simple atelectasis in that pulmonary territory corresponding to the first posterior bronchus was transformed into lobar pneumonia, or more exactly, into a pneumococcic atelectasis because pneumococcic cellulitis developed. This sequence of phenomena was repeated in the second case of Elwyn (46) in exactly the same way.

If in the intrabronchial exudate, instead of pneumococus of low virulence, staphylococci, streptococci, or anaerobes were present, a suppuration or even gangrene of the lung might have ensued. A good illustration of this is offered by the following case of Churchill and Holmes.

In a female, aged 31 years, a currettage, suspension of the uterus, ovariectomy, and appendectomy were done under ether anæsthesia. Immediately after there was dry cough, and later green sputum appeared. About 2 months later the sputum became foul. Fourteen days after the onset she was admitted to the Massachusetts General Hospital presenting a dullness at the base of the right lung below the scapula, diminution of vocal and tactile fremitus and of breath sounds. A few moist râles were present. The sputum was foul; about 7 ounces per day. Bacterial examination showed influenza bacilli and streptococci. Roentgenograms showed the mediastinum displaced to the right and the movements of the diaphragm markedly limited on the right side.

These case histories illustrate the striking similarities between postoperative atelectasis and postoperative pneumonitis. They demonstrate the paramount importance in the various suppurations of the lung, of the obstruction of a bronchus with consequent suppression of the normal drainage of the corresponding bronchial tree; such drainage is the natural mechanism for self defense and sterilization of the lung.

THEORY

From experimental and clinical investigation, the conclusion is drawn that there are no differences between postoperative pneumonia and postoperative atelectasis other than those due to the type and the virulence of the micro-organisms infecting the occluding bronchial mucus. I am convinced that the determining factor in the production of this condition is the more or less temporary "plugging" of a bronchus by mucus, which is followed by the absorption of the alveolar air and atelectasis of the corresponding lung. However, the term "plugged" must not be taken literally—no more so than the term "corking" used by Chevalier Jackson (71). The obstruction of a lung does not depend only upon the consistency and viscosity of the bronchial exudate. It depends as well upon the expelling force of the lung. Very viscid and tenacious mucus may not be able to obstruct a lung which maintains unimpaired its means of defense, namely coughing, respiratory movements, and activity of its ciliated epithelium. The cilia probably help break up the column of mucus, and cough expels this mucus from the bronchus. On the contrary, very thin mucus may be able to obstruct a bronchus when the lungs are at a disadvantage—as they often are after operations because of suppression of the cough reflex by narcotics, pain, posture, splinting of the thorax, paralysis of the respiratory muscles, or because of general weakness in

wasting illnesses, bedridden patients, etc. An example of the possibility of bronchial obstruction by thin mucus is given in the case of Harrington.

A man of 28 years was operated upon for right nephrectomy under ether anæsthesia. Twenty-four hours later typical atelectasis of the left lung appeared and was complete in 24 hours. The left lung was completely opaque, the heart and the trachea displaced to the left, and diaphragm invisible, because of the dense shadow. A bronchoscopic examination was made. Upon the introduction of the tube, a thin serous secretion poured out of the trachea . . . the consistency of the fluid was not thick as in the case observed by Tucker. Dyspnœa and cyanosis subsided immediately after aspiration of the fluid. A roentgenogram taken 15 minutes after showed the heart in normal position and a marked decrease in the pulmonary density.

Pneumococcus is reported practically in every case of atelectasis and postoperative pneumonia in which bacteriological examination has been made. Furthermore, as A. O. Whipple has shown, this pneumococcus, group 4, is identical with pneumococcus isolated from the mouth of the patients previous to operation. This pneumococcus is generally of a low virulence so that the exudate will not be rich in fibrin. Wadsworth has clearly established that the amount of fibrin in the exudate is proportional to the virulence of the organism.

What may be the course of an atelectasis once established? There are various possibilities.

1. During a coughing spell the main column of mucus may be disrupted or expelled and the affected lung is rapidly aerated; or there may be a partial expulsion and only partial aeration of the parenchyma. Another coughing spell will evacuate more mucus, some bronchi will be freed, and so on until the lung is completely aerated. The fact that aeration of the lung proceeds from above downward makes it easy to conceive the "creation of an airway" by disruption of the obstructing mass of mucus and consequent transformation of a complete obstruction into an incomplete one. By a subsequent occlusion of a previously liberated bronchus, an already aerated portion may again be obstructed and become atelectatic. This explains the variability of the physical signs. The rapidity of the expulsion of mucus depends not only upon the condition of the patient and the expelling force of his thorax, but also upon the amount of fibrin present in the exudate—an amount which we have already said was proportionate to the kind and the virulence of the microbes present. The investigation of Archibald and Brown of cough reflex, and the conclusions drawn by Lee, Tucker, and Clerf concerning the production of bronchial obstruction are of great interest in the elucidation of the mechanism of this obstruction.

2. If the obstruction is prolonged, and the virulence of the pneumococci sufficient, a pneumococcic cellulitis (as in medical lobar pneumonia) will follow. This in postoperative cases is generally mild and produces a pneumonitis comparable to the mild type of medical pneumonia (called *maladie de Woillez*). In this way the condition starts as atelectasis and continues to develop as pneumonia (cases of Elwyn). When the major part of the mucus exudate is expelled and only a few bronchi remain occluded, we will have forms similar to those observed by Rigler (Case 5), with pneumococci in the occluded area becoming more virulent.

3. If the obstructing mucus is infected with pyogenic organisms, suppuration may follow, if the obstruction is prolonged. If anaerobes are present, gangrene may ensue. The origin of lung abscesses following tonsillectomies, bronchopneumonia, or pneumonias, are due, I believe, in the great majority, to the same mechanism. The abscesses following "delayed or unresolved pneumonias" are the most characteristic of this group.

Viewed from this angle, the problems of postoperative bronchitis, atelectasis, and pneumonia are greatly simplified. The relation between them has interested several authors. Elwyn (46) suggested that "the greater number of postoperative pneumonias were due to infection taking place in atelectatic or collapsed areas of the lung." In 1924, after a careful study of all operative cases, he says (47): "attention was especially directed toward finding atelectatic areas of the lung in the first few days following the operation. In

many cases following abdominal operations, we were able to observe signs of consolidation over a part of the lobe, which disappeared in 20 to 48 hours, often during the examination, after a fit of coughing. In some of these cases a roentgen-ray examination showed a high position of the diaphragm on the affected side." He considers that atelectatic areas are present most often after abdominal operations and that infection spreads to them due to a bronchitis existing before the operation or caused by the anæsthetic.

It is interesting here to quote Meltzer, who studied the pathogenesis of experimental pneumonia in the dog. He suggests that "in the human a previous cold may furnish a mucus secretion which might occlude several small bronchi and thus prepare a favorable ground for the pneumococci which secondarily infect this mucus secretion; under these circumstances, pneumococci develop rapidly and invade the surrounding tissues. Lee (1924) believed that collapse of the lung in varying degree is a constant phenomenon in any operative procedure and in traumatic or inflammatory injury of the heart and bronchi.

Czerny (34), in 1905, considered as the cause of postoperative pneumonia "retention of bronchial exudate in the bronchi" and created the term "retention pneumonia." The description he gives of it is absolutely identical with that found in "drowned lung" of Leopold (81) in atelectasis.

The relation between pneumonia and atelectasis was seen by these authors; but the nature and importance of this relation had not been grasped. The possibility of secondary infection of an atelectasis, says Elwyn (46), "does not solve the question, but merely puts it back a step further. The question is how does the collapse of the lung arise?" Scott and Joelson (1927), in a rather prophetic statement, have said: "An explanation of the origin of postoperative massive atelectasis will undoubtedly do far more than solve this clinical mystery, since it may prove to be a most important step in reducing the incidence of these fatal postoperative pulmonary complications now classified as pneumonia." But it is really astonishing that the man who clinically discovered massive atelectasis, W. Pasteur (101), had the clearest foresight as to the significance of atelectasis. "I feel sure," he wrote in 1910, "that when true history of postoperative lung complications comes to be written, active collapse will occupy an important position among the determining causes."

I consider that atelectasis in its different forms, multilobar (massive), lobar or lobular (patchy) due to pre-operative or postoperative bronchitis, is not only the forerunner of postoperative pneumonia or bronchopneumonia, but that it is an initial and integral part of the disease syndrome. It is a manifestation of the rôle bronchial obstruction plays in the causation of pulmonary complications. Bronchial obstruction is the starting point of pneumonitis, lobar or lobular, and most probably also of abscess and gangrene as well. The particular condition arising will depend upon the infecting agents. So long as the bronchi are open and their drainage insured, the lung maintains asepsis by the mechanical means at its disposal—evaporation, expectoration, activity of the ciliary epithelium, and the antiseptic power of the mucus (Arloing); but when obstruction occurs, the fate of the parenchyma depends upon the microbes present in the occluding mucus. If they are of low virulence, there will be a slight degree of inflammation, a slight amount of exudate, and little or no fibrin. The air will be absorbed, and the walls of the alveoli will collapse completely, reducing to a minimum the size of the lung with marked displacement of the mediastinum, heart, and trachea, and with elevation of the diaphragm. If the mucus be infected with more virulent pneumococci, then a condition called postoperative pneumonia (postoperative pneumococcic atelectasis), lobar or lobular, will develop. The amount of exudate will be greater and consequently the decrease in the size of the lung less marked and the displacement of the mediastinum less conspicuous. If pyogenic micro-organisms are present (staphylococcus, streptococcus, influenza bacilli, etc.) abscess may result if the occlusion is sufficiently prolonged. If, finally, virulent anaerobes are present, gangrene may ensue.

TREATMENT

By the theory thus developed, the pathogenesis of pathological postoperative pulmonary conditions can be explained and the foundation laid for a preventive and curative treatment, based upon sound etiological principles. Bronchial obstruction cannot be produced unless two factors are present. The first is a more or less viscid bronchial secretion and the second the inability of the lungs to expel it. This second factor will depend upon the viscosity of the exudate and the degree of impairment of the means of defense of the lung. Consequently, the elimination of all or one of the factors named, if this conception be correct, will prevent or cure postoperative complications of the lung (of nonembolic origin). Let us consider each of these factors separately:

1. *Bronchial secretion.* It is known that mucus is abundantly secreted by the innumerable mucous glands of the bronchi (of 1 millimeter diameter and up), as a response to the slightest irritation of the bronchial mucosa, and that it is constantly pushed outward by the ciliary movements. The cilia can propel foreign particles at a rate of 0.5 millimeter per second. Dixon and Inchley described an ingenious apparatus, the "cilioscribe," by which this rate can be measured with precision. Under normal conditions, the amount of mucus secreted is small and the ciliary movement suffices for its elimination. But with the slightest irritation, it increases considerably and its contact in increased amounts with the mucosa of the larger bronchi produces cough which contributes to its expulsion. The expelling force of cough is considerable. For this reason, it is almost impossible to obstruct the bronchi of a dog and produce obstructive lesions if the animal is not completely and deeply anæsthetized, and this for several hours after the obstruction. For that purpose Coryllos and Birnbaum (25–29), and Lee, Clerf, and Tucker used amytal (iso-amyl-ethyl barbituric acid, Lilly) intraperitoneally for anæsthesia in their experimental work. Even then cough was often produced when my obstructing balloon was being inflated in the bronchus, and it was necessary to use a small piano wire spring device in order to avoid expulsion of the balloon. Deep respiration and thorough aeration of the bronchial airways and the alveoli insure against stagnation or accumulation of mucus with possible occlusion of bronchi. The inspiratory dilation of bronchi, the alternating respiratory currents of air, the ciliary movements, and the expulsive powers of coughing are the usual defenses of the lung against infection. To these must be added the antibacterial properties of the bronchial mucus.

But in case of bronchitis there is, besides increased secretion, a number of other modifications, prominent among which are a hyperæmia of the mucosa with a more or less marked degree of œdema and subsequent narrowing of the lumina of the bronchi. Moreover, and this is to my mind the principal factor, bronchial inflammation allows group 4 pneumococci, normally present in the mucosa of the mouth and throat, to descend the air tract; their presence modifies the nature of the bronchial secretion, which now becomes richer in fibrin and much more viscid. A vicious circle is thus created; the increased viscosity renders expulsion of bronchial secretion more difficult, and its prolonged contact with the mucosa increases the inflammatory reaction of this membrane and consequently the amount of bronchial secretion. The motility of the epithelial cilia becomes more and more impaired. If now the cough reflex decreases in force or ceases altogether, if the amplitude of respiration diminishes, and if the patient is immobilized in one position it is easy to understand how bronchial obstruction may occur. The mechanism of cough, described by Archibald and Brown, gives a clear explanation of the production of patchy, lobar, or massive atelectasis, according to whether the obstruction of a small lobar or common bronchus ensues. At the same time it explains how displacement of this mucus can create an airway and result in a temporary or permanent expulsion of the mucus, with a temporary or permanent aeration of the involved lung.

2. *Means of defense of the lung.* The means of defense of the lung have already been mentioned. In postoperative cases, it is easy

to understand that under the combined effect of narcotics, pain, and posture, they are decreased, especially after operation upon the upper abdomen or after thoracic traumatism (J. R. Bradford). Also, as Head and Powers have shown, the vital capacity decreases considerably after operation on the upper abdomen. For the same reasons the lesion appears within 24 hours after operation and clears up as soon as the means of defense of the lung are recovered.

From these considerations, we can conclude that prevention of postoperative lung complications should be possible if we could prevent the formation of mucous exudate and the decreased ventilation of the lung, especially that of the lower lobes. How can this be accomplished? I believe that since the viscosity of exudate is dependent upon the virulence of pneumococcus group 4, every attempt should be made to decrease the virulence of this microbe. Careful pre-operative cleansing of the mouth, a preliminary inspection by the dentist, and a mouth wash with optochin, hydrochloride, 1:500, repeated every 3 or 4 hours, will be of great help. Optochin is a powerful and specific bactericidal against pneumococci. The internal use of optochin base has given some encouraging results (Morgenroth and Levy, A. E. Wright, Baldwin and Rhoades, Walter, Cross). With Dr. J. Cline on the Second Surgical Division of Bellevue Hospital, I experimented with optochin base by mouth and the hydrochlorate salt by rectum, before and for 24 hours after operation without noticing any marked differences with the cases taken as controls. I shall not insist upon the necessity to postpone if possible operation in the presence of a common cold or of a sinusitis. The importance of these conditions for the development of postoperative pulmonary complications has been sufficiently emphasized (Whipple, Cleveland, Churchill, etc.).

I should like to lay emphasis upon another therapeutic agent, which seems to me thus far to exert a marked influence in the prevention of postoperative pulmonary complication. This is the use of a mixture of carbon dioxide and oxygen, or even air. It is not necessary to enter here upon the theoretical or experimental detail of this important question. The results of the experimental work of Henderson and Haggard, Birnbaum and myself will be given in a paper to appear shortly.[1] Suffice it to say that carbon dioxide seems to act in two ways: first, by producing hyperventilation of the lungs, it prevents the deficiency in respiratory excursion which follows operation especially on the abdomen and thus provides the alveoli with the necessary amount of air for expulsion of intrabronchial secretion. Second, it appears from our experimental data that carbon dioxide by decreasing the hydrogen-ion of the exudate acts upon the pneumococcus to inhibit its growth and probably by favoring the proteolysis of the fibrin in the exudate. Both of these actions have as an effect, besides the decrease in virulence of pneumococci, the liquefaction of the exudate and its easier expulsion. Henderson and Haggard (60) in a preliminary paper insisted upon the importance of the last mechanism. The results obtained by Scott and Cutler (1928), Sixe, Dzialoszynski (1927), Fischer (1928), and others show the real importance of this method in prevention of postoperative pulmonary complications.

The authors mentioned used the carbon dioxide-oxygen inhalation with the idea of washing out the ether after anæsthesia, following the advice of Henderson and Haggard (62, 1921) and the brilliant results obtained by these authors in the resuscitation of carbon monoxide asphyxias (61, 1922) and alcohol intoxications (63, 1924). Henderson and Haggard (60) believed that prevention of lobar pneumonia in cases of carbon monoxide asphyxias by treatment of carbon dioxide inhalation was due to the rapid elimination of carbon monoxide. But they could give no clear explanation of the exact mechanism of this action. The experimental work of Coryllos and Birnbaum (27) brought the solution of this problem by showing the relation existing between atelectasis and pneumonia. Hyperventilation acts in both diseases by relieving the obstruction, aerating the apneumatic lung, and above all by reestablishing a free bronchial drainage.

[1] This paper has appeared in Arch. Int. Med., 1930, xlv, 72.

Experimental data to appear shortly will show that in experimental atelectasis in dogs, if we extract the obstructing balloon and place the animal in an atmosphere of air containing 5 to 7 per cent of carbon dioxide, the lung will be aerated very rapidly (15 to 60 minutes). Dogs with experimental pneumonia appear to have a far lower mortality rate if left in this atmosphere for a time varying from 6 to 36 hours.

It is not difficult to understand the aeration of the apneumatic lung after the relief of bronchial obstruction. But it is not easy to understand how pneumonic and consolidated lung will be aerated under carbon dioxide hyperventilation. This supposes the elimination of the occluding agent in pneumonia and it is precisely this mechanism which remains obscure. This point is now under investigation. We can state at present that under the influence of carbon dioxide inhalation the bronchial exudate appears to lose its viscosity, is transformed into a thin frothy secretion, which is more easily expectorated or aspirated by the bronchoscope or resorbed, and the lung can therefore drain and become aerated. In dogs which, after several hours respiration of carbon dioxide 6 per cent air mixture, survive extremely toxic pneumonias, the change was quite rapid; the animals come so rapidly out of the toxic condition that one cannot help being impressed. This experimental work will be given in detail by Birnbaum and myself in collaboration with Henderson and Haggard in a forthcoming paper. At the present time, laboratory and clinical investigations allow us to state that this method constitutes a means of preventing postoperative lung complications far more efficiently than any of the methods thus far employed.

The elimination of other factors favoring development of postoperative lung complications, namely narcotics and posture, should not be neglected. The patient's position in bed should be changed often; if there is no other contra-indication, a moderate Trendelenburg position is desirable. The use of atropine, which very probably after a suppression of secretion renders it more viscid, should be dispensed with.

Once a postoperative lung complication is established, carbon dioxide inhalation is indicated. We should not forget, however, that the liquefied exudate should be expectorated and the method of L. Sante (119), namely, turning the patient on the healthy side several times a day in order to induce cough, must be used. We need not be afraid of the convulsive and the occasional asphyxiating cough which may follow the rolling of the patient upon the healthy side. I have never noticed any untoward effect from such cough. If the condition of the patient is such that he cannot expectorate and if after a reasonable time he does not improve, I consider that there should be no delay in aspirating the bronchial secretion by bronchoscope. We need not fear shock from such a procedure. I have already shown that even in toxic medical pneumonias, there is no shock after a skillfully performed bronchoscopy (24). The number of my cases in which carbon dioxide inhalation has been used as a curative procedure is not as yet large enough to allow definite conclusions to be drawn.

In a recent paper, Binger, Judd, Moore, and Wilder have shown that good results were obtained in the treatment of postoperative pneumonia by the use of oxygen inhalations. Judd and Passalacqua used oxygen inhalations, both as a prophylactic and curative measure. In a group of 180 unselected cases, there was no casualty, although 43 patients already had a slight degree of pulmonary congestion and 32 patients had obvious signs of pulmonary consolidation.

From my own experience I consider the carbon dioxide and oxygen method superior to simple oxygen inhalation. The latter prevents anoxæmia whereas the former, by increasing the ventilation of the lungs, and very possibly by direct action upon pneumococcus and its liquefying effect upon the exudate, strikes at the cause and not only at the symptoms of the disease.

TECHNIQUE

The technique I consider quite efficient is as follows: Immediately at the end of an operation (especially abdominal) and independently of the anæsthesia used, the patient breathes a mixture containing approximately

5 to 10 per cent carbon dioxide and oxygen. If special mixture tubes are not available, a rubber bag is filled with oxygen and carbon dioxide added to it. There is no danger in using mixtures of even 15 per cent carbon dioxide. If a special Henderson and Haggard inhalator (as used by Fire Departments) is available, the valve is fixed at 10 pounds. Immediately after the first few inhalations, respiration is modified and respiratory movements become deeper. After a little experience, it is easy to regulate the amount of carbon dioxide and oxygen given, so as to maintain the patient in a condition of deep breathing. It is not necessary to prolong this inhalation over 3 to 5 minutes at a time. Every 2 to 3 hours, when the patient is in the ward, a tube of the mixture or a bag as described above may be used in conjunction with an anæsthesia mask or a nasal catheter, and carbon dioxide-oxygen is given for 2 minutes at a time. At the same time the patient's position is changed, and this, however severe the operation performed upon him may have been. The more severe and prolonged the operation (particularly if abdominal), the greater is the danger of a postoperative pulmonary complication; this later complication is a far greater danger to the patient than is the change in position. In cases in which consolidation has already developed the use of a tent or chamber with continuous oxygen and intermittent carbon dioxide administration is advisable. Finally in the cases of delayed atelectasis or when the lung appears drowned in its secretions and when cough and expectoration are neither present nor can be induced by rolling the patient from side to side, bronchoscopy should be used. It is a bold measure, at least it is considered such at the present time, but it may be a life-saving procedure.

If the theory that postoperative bronchitis, atelectasis, bronchopneumonia, and pneumonia are simply different stages or manifestations of the same morbid conditions be correct, then the treatment proposed to overcome bronchial occlusion and insure free drainage of the bronchial tree for 48 hours after operation (when the means of defense of the lung are impaired) will enable us to avoid postoperative pulmonary complications. In cases in which these are already developed, the treatment described, by aiming at the cause of the complication, the pneumococcic (group 4) bronchitis, will help us avoid their extension and hasten recovery.

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EXPERIENCE WITH SODIUM AMYTAL AS AN INTRAVENOUS ANÆSTHETIC[1]

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ALTHOUGH for years the barbituric acid derivatives have been used for hypnotic purposes, it has only been since the preparation of the *sodium salts* of these derivatives that they have been applied in surgical anæsthesia. The sodium salts are soluble and are tolerated in doses sufficiently large to be effective.

HISTORY

In 1924, Fredet in France reported the induction of general anæsthesia by the intravenous administration of somnifene, and in 1927 Bumm, in Germany, reported the similar use of pernokton, both barbituric derivatives. In this country in 1926 Page and Coryllos, by preparing the soluble sodium salt of iso-amyl-ethyl-barbituric acid, were able to inject dogs intravenously and intramuscularly to produce anæsthetic relaxation. The dogs went "quietly and rapidly to sleep and awoke after a number of hours active and frisky, without nausea or vomiting." Page and Swanson showed iso-amyl-ethyl-barbituric acid, or amytal, to be more effective and less toxic than barbital (veronal). Subsequently Chambers, Milhorat, Hines, and others have studied the effect of sodium iso-amyl-ethyl-barbiturate upon animal metabolism when given in anæsthetic doses, and their observations are to be found in the literature of the past 4 years.

In February of this year Zerfas and McCallum, of the Medical Research Department of the Indianapolis City Hospital, reported the successful induction with sodium iso-amyl-ethyl-barbiturate of general anæsthesia in man in about 300 cases, and Lundy, of the Mayo Foundation, reported in September at the Pan-Pacific Surgical Congress 1,000 cases, surgical and medical, in which this barbituric acid derivative had proved of value. Of these latter cases in the surgical series, however, 450 were hæmorrhoidectomies, in which the acid amytal tablet was given by mouth.

In this paper we wish to report our experience with sodium iso-amyl-ethyl-barbiturate administered to 195 patients.

Amytal is the trade name of iso-amyl-ethyl-barbituric acid. It is marketed in grain and one-half tablets for soporific purposes. The sodium salt, on the other hand, i.e., sodium amytal, which is soluble, and with which the following report is concerned, is supplied in pure form, as a crystalline powder with companion ampules of distilled water. When ready for injection, the salt is dissolved in the distilled water to give a 10 per cent solution at a hydrogen-ion concentration of 9.8. For convenience in this report the sodium-iso-amyl-ethyl-barbiturate will be referred to as sodium amytal.

DOSAGE AND ADMINISTRATION

The dosage is still in the experimental stage. The lethal dose for man is unknown. As with all hypnotics, there is an individual susceptibility factor. This, to us, seems of greater importance than the weight of the patient in determining the amount the individual should receive. Susceptibility can best be judged, just as in giving inhalation anæsthetics, by the color, blood pressure, pulse, and respiration changes as the drug is administered. Age and general strength are important considerations. Very old or debilitated patients may fall fast asleep after 3 to 4 grains have been given, while young robust patients may require 7 to 9 grains before they lose consciousness. As a general guide, in surgical cases we customarily administer double the amount necessary to put the patient barely to sleep. We no longer give over 22 grains to any patient at one time. This maximum dose may be used for surgery about the face, neck, or breasts, where for convenience or other reasons it is desired not to use supplementary anæsthetic agents. Where it is desired to produce only a twilight stupor and retain the cooperation of the patient, as in regional and

[1]Read before the Washington State Medical Association, Yakima, Washington, August 29–31, 1929; before the King County Medical Society Seattle, Wash., November 18, 1929; and before the Thirty-Ninth Annual Meeting of the Western Surgical Association, Del Monte, Calif., December 12–14, 1929.

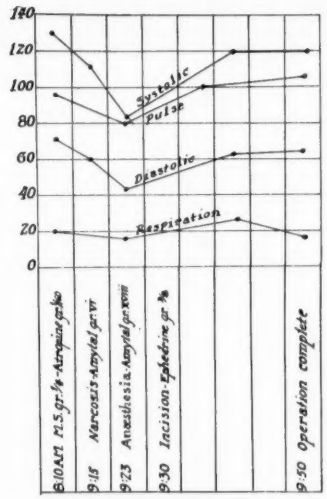

Chart 1. Representative curve of blood pressure, pulse, and respiration. Case 12. Appendectomy. Woman, aged 63 years. In this case no supplementary anæsthesia was used.

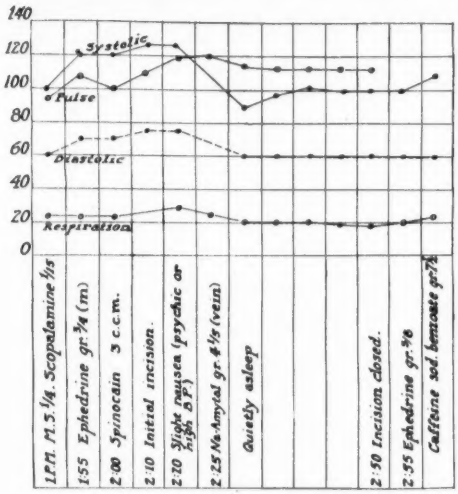

Chart 2. Combined spinal anæsthesia and sodium amytal. Case 124. Subtotal hysterectomy. Woman, aged 43 years. No inhalation anæsthetic necessary. Condition good. Convalescence very pleasant.

spinal anæsthesia, smaller amounts must be given, and in these cases it is best to administer the drug by vein because the more rapid action permits more accurate control of the depth of unconsciousness.

Sodium amytal, as described, is soluble and it may be administered orally, intravenously, intramuscularly, or by rectum. In all but two of the surgical cases listed in this report, the administration was by vein. By this route the 10 per cent solution at a hydrogen-ion concentration of 9.8 should be injected at a rate not exceeding 1 cubic centimeter per minute; the action is immediate. When given intramuscularly the induction is delayed 10 to 40 minutes, but the effect is more lasting. We have never administered the drug intraperitoneally. In one case of intravenous administration, where the solution was spilled into the tissues, a very sore arm resulted, so that we have not given any subcutaneously. By mouth and by rectum larger dosage is required.

ACTION

The exact localizing action of the barbiturates is not known. As Isenberger points out, they probably act by depressing certain vegetative centers in the hypothalmic portion of the diencephalon, but this is not established—any more than is the physiology of sleep.

INDUCTION

The induction of sleep is rapid and quiet, almost dramatic. During the intravenous administration of the first 3 to 9 grains the patient may remark that he is feeling sleepy, and if engaged in conversation he begins to slur his words; finally he may yawn, and drop off into a quiet sleep in the middle of a sentence, while the drug is still being given. As contrasted with inhalation anæsthetics, we have observed an excitement stage in only one case, that of a chronic alcoholic, who sang vulgar songs for several minutes before losing consciousness. Several others experienced brief coughing spells during the induction.

While with 3 to 9 grains the patient is asleep, with a few grains more he seems to be reflexly hypersensitive, squirming, though asleep, to the slightest needle prick; thereafter, as the dose is increased, more profound anæsthesia is produced.

REFLEX, BLOOD PRESSURE, AND PULSE CHANGES

The pupils become contracted and in some cases fixed so that they will not react to light.

TABLE I.—SUPPLEMENTARY ANÆSTHESIA USED

	Cases
N_2O-O_2 (85% to 50%)	27
N_2O-O_2 and ether	56
Local procain	10
Local procain and N_2O-O_2	16
Spinocain—spinal anæsthesia	12
Ether	17
Total	138
Sodium amytal alone	27
Total	165

This *fixation* of the pupils occurs in greater frequency where inhalation anæsthetics or large doses of morphine have been superimposed. The corneal wink reflexes are diminished. The gag reflex is usually present. The knee jerks are often exaggerated during the period of hyperæsthesia, and they have still been elicited with the deeper degrees of anæsthesia we have secured by our larger doses. This preservation in some cases of the reflex responsiveness with sodium amytal is held as an advantage since it is theorized that the patient can better react to stimulation. On the other hand the persistence of these reflexes may tend to disprove the induction of complete anæsthesia with protection against surgical shock to the nervous system.

The blood pressure in all but a few of our series fell, the systolic an average of 30 millimeters of mercury, the diastolic an average of 15 millimeters, during the induction of anæsthesia, but returned to the normal level early in the operation or else was restored to normal by the administration of ephedrin. In one patient with hypertension and heart block, undergoing an operation for toxic adenoma of the thyroid, the systolic pressure fell 100 millimeters of mercury during the administration of the first seven grains of the drug (Charts 1 and 2).

The pulse rate, if elevated by emotional excitement, was reduced during the induction of anæsthesia. In all other cases, however, the pulse appreciably quickened after injection of the sodium amytal, an average of plus 15 beats per minute. The respirations become shallow and the respiratory rate slightly increased, but in a few cases the respiratory rate fell as low as 12 per minute. The color in all but a few cases has been uniformly good.

PRELIMINARY MEDICATION

In all the surgical cases reported we have given morphine sulphate 1/16 to 1/4 grain with atropin sulphate 1/150 grain one hour prior to the administration of the sodium amytal. In one-half of the surgical cases we have followed Lundy's regimen of administering chloretone 10 to 12 grains 1½ hours beforehand. In the cases receiving the preliminary chloretone, sleep was induced with an average of a half grain less of sodium amytal, and the hyperæsthesia seemed to be less marked and of shorter duration. However, the reaction time was neither lengthened nor shortened by chloretone and the incidence of restless reactions was not reduced. We are therefore beginning to question the justification of adding chloretone to the already complex combination of hypnotic agents.

SUPPLEMENTARY ANÆSTHETIC AGENTS

When we first started to use the drug we knew little about it, and from the overenthusiasm of the earlier reports we were led to expect surgical anæsthesia unassisted by other anæsthetics. We soon proved to ourselves that this was inadvisable. As described above, the patient becomes hypersensitive after a small dose of the drug, then more relaxed as the dosage is increased. But with as large dosage as we feel it wise to use, we have in only 4 cases obtained satisfactory relaxation for abdominal surgery under sodium amytal alone. Two of these were women past 60 years of age, and the other two, while younger, were of asthenic stature and debilitated by chronic illness.

The types of supplementary anæsthetics selected are outlined in Table I. In all thyroidectomy cases receiving sodium amytal we have given smaller dosage, 8 to 15 grains, in order that the patient might be made to strain and speak at the end of the operation to permit early recognition of hæmorrhage or injury to the laryngeal nerves. Hence it had to be supplemented with either local novocain infiltration or light nitrous oxide and oxygen inhalations. For abdominal surgery nitrous oxide and oxygen had to be superimposed in some cases because of the squirming hypersensitiveness of the patient, and in other cases

along with a small amount of ether vapor in order to get sufficient relaxation. In these cases the dosage of sodium amytal has varied from 12 to 22 grains—except in a few very susceptible patients when we first started to use sodium amytal, where the deep cyanosis caused us to cut the dose very low. Since we see but few instances of this initial cyanosis now, we believe it to have been due in these early cases to relaxation and swallowing of the tongue or to an overdose of morphine beforehand.

Probably the most satisfactory combination to both surgeon and patient is that of *spinal anæsthesia* and sodium amytal, the spinal anæsthesia relaxing the muscles ideally for the surgeon, the sodium barbiturate making the patient oblivious of the proceedings in the operating room and adding to his postoperative comfort. Unless thoroughly familiar with both sodium amytal and spinal anæsthetics the anæsthetist may find it easier first to establish the level of anæsthesia with the spinal anæsthetic and thereafter administer the sodium amytal, rather than the reverse procedure. For when the sodium amytal is given first the patient often proves hypersensitive and this makes the lumbar puncture difficult. Furthermore in using Pitkin's light spinocain the hypersensitiveness necessitates constant guard afterward to prevent the patient from raising his head with consequent danger of respiratory paralysis from ascent of the light spinocain to the medulla. However, with studied judgment, the most satisfactory of all results may be obtained by administering in the room beforehand sufficient sodium amytal to put the patient barely in a twilight stupor, such that he is free of anxiety yet can still be aroused to co-operate for the lumbar puncture. Later, after the spinal anæsthetic has set, more of the sodium amytal may be given to put the patient entirely to sleep (Table II).

In our endeavor to give the new drug with anæsthetic properties a fair experimental trial, the surgeon has often had to work at a disadvantage because we have withheld auxiliary agents until they were absolutely necessary. In a number of cases the incision has been made and the peritoneum reached under sodium amytal alone, only to find it necessary to hold up the operation until sufficient relaxation could be obtained with gas and ether. It is necessary, therefore, to understand the limitations of the drug, and to supplement it at the proper time with the proper anæsthetic.

TABLE II.—REGION OR TYPE OF OPERATION

	Cases
Breast	16
Thyroid	23
Nose	4
Face	2
Eye	2
Mastoid	3
Mouth (tongue, jaw, teeth)	6
Hand (amputation)	1
Glands of neck	1
Gall-bladder and bile ducts	15
Kidney	6
Shoulder	3
Laparotomy (inoperable cancer)	3
Appendix	16
Stomach, duodenum	3
Small intestine (anastomosis)	5
Cæcum, colon, rectum	12
Pelvis (uterus, tubes, ovaries)	27
Perineum	9
Hernia	3
Varicocele	2
Cystoscopy	1
Spleen	1
Parathyroid	1
Total	165
Medical cases	21
Obstetrical cases	7
Cæsarean sections	2
Total	195

This necessitates that the anæsthetist become familiar with the new combination of anæsthetics. Otherwise many exasperating and occasionally dangerous conditions may occur. The anæsthetist must become accustomed to the changes in the pupillary reactions, in the pulse rate, and in the general muscular tone. New guides to the depth of anæsthesia must be established. In several instances the fixation of the pupils, by preventing their dilatation with the dangerous depth of anæsthesia, permitted the supplementary anæsthetic to be pushed to the point of temporary respiratory cessation before the anæsthetist realized that too much ether had been given. In these cases a little oxygen and a few strokes of artificial respiration restored the patient.

ADVANTAGES

The chief advantage of sodium amytal in surgical management is the way in which it

TABLE III.—POSTOPERATIVE COMPLICATIONS IN ONE-HUNDRED-FIFTY SURGICAL CASES

	Cases
Nausea	4
Vomiting	14
Headache (all relieved by aspirin)	5
Restless reaction	10
"Drunken" delirium (13 hours after sodium amytal)	1
Hysterical psychosis 1 to 3 days	3
Deep cyanosis (swallowed tongue)	1
Catheterization necessary up to fifth day after operation	3
Catheterization necessary only 1 to 2 times	45
Involuntary urination	1
Bronchitis	1
Pulmonary œdema	2
Backache	4
Sore arm (from injection)	1
Skin rash	1
Respiratory difficulty on operating table	3

spares the feeling and nerves of the patient. He is put to sleep in his room, remembers nothing of the dreaded trip to the operating room, reacts partially 2 to 13 hours after the so-called "hypodermic," remains in a twilight stupor for 24 to 48 hours longer, and in the majority of cases experiences no nausea or vomiting.

Some of the patients who received sodium amytal had had previous operations under inhalation anæsthetics, and to hear their praise of the "new anæsthetic" makes one appreciate the real anxiety some patients suffer before going to the operating room. All patients in the series were asked before leaving the hospital for their opinion of the sodium barbiturate, and every one of them, even those with restless reactions, was grateful for its administration, and many, especially those having been nauseated by ether at former operations, were enthusiastic in praise. Several patients requiring a second operation *requested* the sodium amytal.

Among its other advantages, only less important, is the fact that the amount of inhalation anæsthetic is reduced by one-fourth or more, the depth of ether anæsthesia is more constant, and if nitrous oxide and oxygen are used, the increased proportion of oxygen gives the patient a better color. In waiting for pathological reports on frozen sections, the supplementary inhalation anæsthetics may be temporarily discontinued without the patient arousing. It has been brought out that the sodium barbiturates successfully control the convulsions, vasomotor disturbances, and respiratory difficulty of novocain poisoning, and this is another advantage in its combination with local anæsthesia, where every now and then such a reaction has occurred. In breast amputations the shallow respirations facilitate surgery and may serve to diminish bleeding. In using the live cautery about the tongue and face, we have gotten sufficient relaxation without the use of other anæsthetics.

DISADVANTAGES AND COMPLICATIONS

Some patients having received the larger dosage become very restless and thrash about the bed, and react in a drunken delirium. (This is not true, however, when the drug is given intravenously in small doses.) In the average case of this kind the early administration of morphine and catheterization of the bladder will prove effective. (The bladder loses its tone and cannot empty itself.) Other patients lie motionless so long that there is danger of passive congestion and with the shallow respiration, possibility of pulmonary œdema. Pulmonary œdema is the chief complication to be feared. Of two instances in our series, one was fatal. This represents the only death in the series in part traceable to sodium amytal. The patient was an old luetic man, very toxic and debilitated, undergoing an exploratory operation for obscure abdominal cancer. He received 6½ grains of sodium amytal followed by drop ether. Postoperative pneumonia followed within 12 hours, and the patient died 8 hours after the onset, showing no resistance to the infection. Of course, a case of this type is always a bad ether risk, and we cannot say that he would not have contracted postoperative pneumonia without the sodium amytal. However, sodium amytal *does* depress the respiratory center, and several cases of pulmonary œdema are already on record. It is evident that care should be exercised in the administration of ether to patients who have received sodium amytal and particularly in the case of the aged and debilitated. The other case of congestion cleared rapidly with the use of the oxygen tent and caffeine and promotion of deep breathing on the part of the patient. It should be recalled here that hyperventilation of the

lungs at the end of the operation with the aid of carbon-dioxid-oxygen inhalations is advantageous regardless of the anæsthetic used.

Other complications are listed in Table III. Of the 14 cases in the series with postoperative vomiting, 4 were patients suffering from gall-bladder colic, in whom pre-operative nausea had been even more marked than was the postoperative nausea, 4 others had had extensive pelvic operations, another was a patient with peritonitis with distention, another had undergone both a cholecystectomy and a Judd operation for peptic ulcer—10 cases in which vomiting was rather to be expected. Of the 4 remaining, 2 had had a thyroidectomy, 1 a radical breast amputation, and the last the removal of a stone from the kidney pelvis. All of these except the 2 patients undergoing thyroidectomy and the 1 having the breast amputation received supplementary ether.

The high percentage of catheterizations is deserving of comment. Fifty-seven per cent of the females had to be catheterized as against 23 per cent of the males. This may be explained in part by the fact that we have a standing order on the wards for female patients receiving sodium amytal to be catheterized at the end of 12 hours if they have not voided—but for males only if in pain. This is necessary because of restlessness with the relaxed bladder, which cannot empty itself. Three cases of cystitis have resulted with this atonicity of the bladder and catheterization.

In relaxed cases the jaw may droop and the patient be suffocated from swallowing his tongue. So grave a calamity from this source almost befell one of our patients that we now keep a special nurse at the bedside until the patient is fully conscious.

LABORATORY FINDINGS

The following laboratory findings are merely a preliminary report and cannot be interpreted as representative of sodium amytal alone, because supplementary anæsthetics were superimposed upon the sodium amytal in the majority of cases, and because we have no control series of similar tests on patients undergoing operations under other anæsthetics. Before they can be so interpreted similar tests must be correlated to rule out the separate influence of surgical shock and of the auxiliary agents used.

In 77 patients the average output of urine for the first 12 hours following operation (calculated from the nearest voiding time) was 315 cubic centimeters; for the first 18 hours 430 cubic centimeters; and in 21 recorded cases for the first 24 hours 630 cubic centimeters. In 72 patients the specific gravity of the first urine voided after the sodium amytal averaged 1.011 as against an average of 1.006 for the morning urine voided before sodium amytal. Alkaline urines became acid after the drug, and urines initially acid remained acid. As pointed out by Zerfas, the excretion of urates in the urine is increased. Repeated urinalyses at varying intervals after the drug showed no albumin, casts, or red blood cells.

In 22 patients tested, the average blood urea and creatinin immediately after and 24 hours after the operation under combined sodium amytal and ether anæsthesia showed a slight increase over the fasting level before operation.

In 36 patients the average blood sugar taken immediately after operation under combined sodium amytal and inhalation anæsthesia showed an average increase of 26 milligrams per 100 cubic centimeters over the fasting level before the operation, and an average increase of 27 milligrams per 100 cubic centimeters when taken 24 hours after the sodium amytal. (This series excludes those patients receiving glucose intravenously.) Fifteen of 65 postoperative urines tested gave a positive sugar test. Since we have no blood sugar curves and urine analyses on control cases undergoing operation without sodium amytal and with other anæsthetics used alone, these tests are not conclusive, but they do favor support of the findings of Hines, Boyd, and Resse. These authors, working with dogs, concluded that sodium amytal interfered somewhat with the glycogenic function of the liver—a point to be remembered in administering the drug.

Red blood counts and hæmoglobin readings taken on 65 surgical patients (a) just before, (b) immediately after, (c) 24 hours after, and (d) 10 days after sodium amytal demonstrated no change, except a lowering in two cases complicated by secondary hæmorrhage. Spectroscopic examination was not performed.

TABLE IV.—MEDICAL CASES

	Cases
Pernicious vomiting of pregnancy	1
Coronary occlusion	2
Renal colic	2
Eclampsia	2
Tetanus	2
Pelvic pain (ca. cervix)	1
Chronic alcoholics	3
Traumatic hysterics	1
Hyperthyroid psychosis	1
Insanity	1
Resistant insomnia	3
Hysterical vomiting	1
Morphine addict	1
Total	21

While these results are not conclusive in a positive way they do tend to support the relative safety of the drug.

MEDICAL CASES

In application to medical cases, as a pure and rapidly acting hypnotic, we believe the sodium barbiturates will prove of greater advantage than in surgery. With Doctors Palmer and Blackford we have studied the use of sodium amytal in 21 cases, as listed in Table IV. Some of these received the drug by vein, others by mouth, and a few by rectum. In some of the cases we repeated the administration every 24 to 48 hours over several weeks time, with no apparent complications, and without the patient acquiring a tolerance for the drug.

The case of pernicious vomiting of pregnancy had not improved with isolation, glucose injections, and sedatives. She was relieved for 2 days by the first sodium amytal injection (5 grains). Recurrences of vomiting necessitated repetition of the drug six times over 18 days before the patient was discharged on a full diet. She was a very grateful patient.

A patient with coronary occlusion, who had gotten no relief from 2 grains of morphine, was relieved of the agonizing chest pain by 5 hours' sleep with sodium amytal, awaking without any pain. A second case seen, not in the throes of the anginal attack but later with marked orthopnœa and insomnia, was also grateful to the barbiturate for several nights' sleep.

In the case of a primipara at term with rapidly recurring eclamptic convulsions two doses of sodium amytal (each 4 grains) controlled the convulsions. At the time of the first injection pains were fairly strong and were coming every 5 minutes, the cervix was partially effaced but not dilated, and the fetal heart could not be heard. Four hours later a stillborn babe with rigid extremities, apparently dead some hours, was delivered. One hour after delivery the patient was thrashing about the bed, decidedly on the verge of another convulsion, so that although the pulse was weak and the blood pressure could not be read, another dose of sodium amytal was given. The patient was quiet after this, and the blood pressure, pulse, and color improved with rest. However, 18 hours later the pulse became imperceptible and the patient expired.

A second patient experiencing several eclamptic convulsions within 12 hours following delivery of her baby had the convulsions controlled by 10 grains of sodium amytal intravenously. She made a recovery.

We have administered sodium amytal intravenously to 2 patients with tetanus. The first, a man in dire extremity, with continuous convulsions, died, although the one injection of the sedative controlled the convulsions. The second, a boy 12 years of age, admitted to the hospital in his first tetanic convulsion following 21 days after running a nail in his foot, had the convulsions controlled by 5 grains of sodium amytal intravenously. The dosage was repeated in 2 hours, and several additional times the next 4 days. Meanwhile large amounts of tetanus antitoxin were administered intravenously and intraspinally while the patient slept. This patient made a recovery and on leaving the hospital remembered nothing of his numerous treatments.

In two of the alcoholics we found large amounts of sodium amytal necessary to control the patient and that even then the sleep was only of 4 to 6 hours' duration and the reaction violent. In the third alcoholic it produced a long quiet sleep. As an aid in the Townes-Lambert treatment of one morphine addict, it proved of no definite advantage. The patient slept 8 hours after 22½ grains of sodium amytal by mouth and 7 hours after repetition of the dose—the morphine having been reduced over a 4 day period before this from 5 grains daily to nil. But the reaction

was restless and the weakness and tremors consequent to the reduction of morphine did not seem to be helped by the drug.

OBSTETRICAL CASES

In conjunction with Doctor Houston, Doctor Windom, and others of the hospital's obstetrical staff we are beginning to employ the drug in obstetrical cases, but at present our series is limited to 7 cases, and we can give no opinion of value.

CONCLUSIONS

Finally, our present impressions concerning sodium iso-amyl-ethyl-barbiturate in surgical cases are as follows: We have come to employ it as a rapidly acting hypnotic rather than as an anæsthetic to take the place of ether, nitrous-oxide, novocain, etc. In operations about the nose and in using the cautery about the mouth and neck and in the removal of breasts with the cautery, we believe it of singular advantage, and, as mentioned, in these cases supplementary anæsthetics have not been necessary, and sodium iso-amyl-ethyl-barbiturate has served as a complete anæsthetic in itself. But to look upon sodium iso-amyl-ethyl-barbiturate primarily as a hypnotic rather than as an anæsthetic is not to underestimate the advantages of the drug or its contribution to the comfort of the surgical patient. In highly strung patients psychic shock can equal surgical shock, and if by putting such a patient knowingly or unsuspectingly to sleep in his room we can reduce this type of shock we have added materially to surgical management. We believe that by doing away with the anxiety of the trip to the operating room and the psychic suffocation of the ether mask, as much as by adding to the comfort of the patient after operation the sodium barbiturates have won a lasting place in anæsthesia.

In the sodium salts of the barbituric acid series the internist has a more dependable hypnotic and a rapidly acting one, as well as a control for eclamptic, tetanic, and strychnine convulsions. These derivative salts will prove a great asset in relieving pain and insomnia not responding to ordinary measures.

We feel that there is no contra-indication to the use of this drug where surgical interference is necessary except in case of extreme shock, uræmic coma, diabetes, or respiratory obstruction. It should be given cautiously to the very aged and to those very susceptible to morphine.

We wish to thank Drs. G. M. James, H. R. Wesson, and A. L. Carter for their assistance in administering the drug.

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DIVERTICULITIS OF THE COLON[1]

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DIVERTICULOSIS, or sacculations along the lumen of the large bowel, is a condition uniformly asymptomatic save in a small number of cases in which inflammatory reactions are taking place secondary to the irritating processes. Because of obstruction, or for other reasons, fæcaliths may not be discharged into the colon, but may remain imprisoned, to cause a condition no longer recognized as unique, namely, diverticulitis.

Anatomically, the acquired diverticulum or diverticula, for they are in most instances multiple, and may be localized or scattered diffusely throughout the whole length of the large bowel or even throughout the entire gastro-intestinal tract, represent either a muscular defect or a protrusion of the mucosa through an attenuated spot in the adjacent underlying coats so that there are only two layers in its wall. These two layers are the peritoneum and mucosa, except in the occasional instance in which a true pouch, with all the normal intestinal coats, develops in adult life and possesses greatly thinned-out coats.

Klebs, in 1869, directed attention to the etiological relationship of diverticula to the blood vessels in the intestinal wall. Preceding this contribution, Virchow, in 1853, is credited with having described certain inflammatory areas at the hepatic, splenic, and sigmoid flexures of the colon, which he termed "isolated circumscribed adhesive peritonitis." His consideration of their sequelæ explained the symptoms, although he could not draw a clinical picture of the conditions; he believed that the symptoms usually were interpreted as relating to an inflammation in some other viscus. Graser, in 1898, and Fischer, in 1899, again added to the knowledge concerning this condition from material obtained at necropsy. Graser, particularly, emphasized the predominant occurrence of diverticulosis in the pelvic colon and called attention to the venous congestion without inflammation which occurred in such cases, a view which later experimentation apparently has negatived.

ETIOLOGY

The cause and method of production of diverticula continue to be controversial subjects, but the factors involved in the inflammatory processes of diverticulitis are most readily explained by obstruction at the neck of the diverticulum and its concomitant failure to empty. Whether or not the majority of diverticula are of the congenital or of the acquired type is not clear, and it seems that they may be designated as true or false just as accurately from the anatomical standpoint. When the sacculation is composed of all of the structures normally found in the wall of the bowel, arranged in normal sequence, true diverticula are the result; such are typified by Meckel's diverticulum or by the vermiform appendix, which is a normal vestigial pouch. Rarely, a diverticulum similar in structure to the true type may develop during the patient's lifetime, thus precluding its classification as congenital, but it is usually the result of traction, as reports of cases by Neumann, Nauwerck and Hansemann would indicate. Their rarity makes unnecessary their inclusion in any general classification of diverticula either from the anatomical or the etiological standpoint. Those of the acquired or false type, which occur throughout the gastro-intestinal tract, may be satisfactorily nominated from the etiological standpoint as either "pulsion" or "traction" diverticula. Concerning the traction variety, first described by Rokitansky, in 1861, and most commonly associated with the small intestine, Klebs deserves the credit for the suggestion that the pull on the bowel, by the mesentery, produces the necessary place of lessened resistance. It is con-

[1]Read before the Interstate Postgraduate Medical Assembly of North America, Detroit, Michigan, October 23 to 25, 1929.

ceivable that a diverticulum which contains all of the anatomical structures of the intestinal wall may form in youth or adult life and that subsequently, because of some muscular defect or inherent weakness, the mucosa protrudes between the muscle fibers and an acquired diverticulum results. Observers are not in complete agreement as to the exact situation of these diverticula in the circumference of the bowel. A cursory examination of the literature reveals wide divergence of opinion as to whether the diverticula occur more particularly in the weakest spot in the intestinal wall, which is opposite the mesenteric border, or whether they are associated more often with openings between blood vessels which come in from the mesenteric side. In the small intestine, acquired diverticula usually are constant in their relationship to the wall of the bowel, developing at the mesenteric attachment or a little above it. Usually they attain the size of about 1 centimeter in diameter, but occasionally they become many times larger. In the colon, on the other hand, diverticula are not constant in their relation to the mesentery, but may be found at any point along the circumference of the wall of the bowel, a fact which has added to the confusion as to etiology, particularly as it is affected by the relationship of the blood vessels and the longitudinal muscle bands. Keith is the chief advocate of the view that intracolonic pressure, as evidenced by contracture of the tæniæ in the segment of bowel that is host to the diverticula, is the foremost etiological factor. His contention that this results in the formation of circular folds of mucous membrane in the colon, producing obstruction, and resulting in sacculations in the weak spots of the musculature, is not without other advocates; certainly it must be considered among the more satisfactory explanations of the mechanism of production of diverticula. The structural and anatomical influences in the formation of diverticula have received much attention, particularly the relationship of the blood vessels and the longitudinal muscle bands. Drummond called attention to these influences, and his observation that diverticula occur most commonly between the mesentery and longitudinal bands, and that after penetrating the muscular coats they follow the shields of the mesentery, has been our clinical experience.

The experimental production of diverticula is extremely difficult because of the conditions under which attempts must be made to reconstruct normal factors as they are in the viable bowel. Chlumsky, Philipowicz, Beer, and others have demonstrated by experiments on intestines of dogs that when injections are made into the small bowel while it still is viable, and before it has been removed from the animal, rupture occurs opposite the mesentery. Just the reverse is true when the bowel has been removed from animals and has been dead for a number of hours; then rupture is into the mesenteric portion. The spaces around the blood vessels, as they enter the mesenteric border of the bowel, are empty and non-elastic. Consequently, they do not resist intracolonic pressure in the dead bowel, and their distention by injection of water is not comparable to normal distention; a dead bowel may not be dilated to the size one frequently finds clinically in volvulus. Furthermore, experiments of these same workers have demonstrated that the tear in cases of ileus usually is from the serosal side toward the mucosa. All of this evidence negatives the theory that the weakest portion of the wall of the bowel is in the mesentery and demonstrates conclusively that just the reverse is true; namely, that the point of least resistance is opposite the mesenteric border. Beer, in his experiments in 1904, refuted many of the hypotheses already advanced as to the production of diverticula and the relationship of constipation, venous stasis, and so forth, to their formation. He emphasized the fact that the supposed weakness at the mesenteric border does not exist and consequently cannot account for the production of all diverticula. He stated that there is some change in the resistant power of the intestinal wall, and that there is consequent muscular deficiency which probably accounts for the formation of false diverticula. Indeed, this hypothesis must be looked on favorably, since it will explain the production of diverticula in both mesenteric and non-mesenteric portions of the bowel. According to his hypothesis, diverticula would

develop where the muscular weakness is localized to a small area of muscular tissue; the mucosa would be pushed along the lines of least resistance, the weakened muscle bundles parting, and the direction of the sacculation would be the course of least resistance on the mesenteric sides along the veins. He further stated that larger diverticula, likewise, could be explained in this manner, since larger areas of the musculature could be weakened and could allow the passage of the walls into a sac.

That no one factor produces diverticula seems the most likely conclusion of the study of the experiences of the many observers. It seems reasonable to assume, however, that the outstanding features of their formation have to do with inherent weakness of the wall of the bowel, in addition to increased intracolonic pressure, which results from constitutional or environmental causes. Certainly there must be some congenital predisposition in many cases, and undoubtedly obesity, venous stasis, and constipation, with their noxious cycle of intoxication and lowered resistance, play a part. To this one might add the questionable promoting factor of retrograde peristalsis, but with the mental reservation that there is little conclusive proof as to its actual, positive influence. Once the diverticulum is formed it becomes a bottle-shaped process, with a narrow mouth and wide body, into which the fæcal current projects itself and from which it is released reluctantly. There is consequent inflammatory change secondary to obstruction and stagnation and the rather constant pathological picture.

PATHOLOGY

The pathological processes which occur in the diverticula produce inflammatory changes, with complications of perforation, stricture, and fistula in some cases. At first these changes are local, and, as they progress, they often become extensive and complicated. One should recall, however, that in most instances diverticulitis does not develop on a basis of diverticulosis and that often, when it does develop, it is more likely than not to run a chronic uncomplicated course. Most often, the pathological changes which require intervention or treatment occur in the pelvic portion of the colon and in the rectum, probably because of the stasis which normally the fæcal current undergoes in this portion of the large bowel. In addition, the character of the intestinal content, which is formed and hard, tends to prevent emptying of the diverticulum, once it is packed; the right side of the colon and the small bowel, normally housing fluid content, rarely are subject to impaction, obstruction, and inflammation. The local inflammatory changes begin, of course, in the mucous membrane of the diverticulum, which undergoes atrophy, with subsequent round cell infiltration of the submucous coats, and lateral ulceration. These changes progress both in the wall of the bowel and in the mesentery itself, producing symptoms of inflammation or obstruction, according to the amount of encroachment on the lumen from the inflammation, perisigmoiditis, and mesenteritis. The changes in the diverticulum itself are aptly noted by Wilson in a description of a resected specimen, as follows: "The walls of the diverticulum consist of the following coats: (1) mucosa, markedly atrophied around the proximal end of the lumen where pressure has been greatest from the thickened gut walls, and fairly well preserved around the saccular portion; (2) submucosa, a strong fibrous coat, thicker in the proximal than distal portion; (3) muscularis, fibers derived from the circular coat of the sigmoid, and thickened by fibrous infiltration to twice the thickness of the same coat in the normal portion of the sigmoid; the muscularis in the wall, where penetrated by the diverticulum, was much thicker; and (4) a layer of fibrous tissue from the subserosa."

The changes in the coats of the diverticulum vary markedly from attenuation of the musculature to complete absence of musculature; in the latter condition, the wall is made up mostly of mucosa and serosa. The chronic thickening of the mesentery, so frequently noted in diverticulitis, is the result of extension of an inflammatory process and at times of perforation into the mesentery. Usually there is marked thickening of the mesenteric attachment, as well as contraction, and often considerable tumefaction. Perforation of the diverticulum may produce local ab-

scess formation or fistula, external or internal. It is not uncommon for a vesico-intestinal fistula to result from diverticulitis after an abscess has been formed in the pelvis, or after the thickened sigmoid has become attached to the bladder and the process has extended slowly by necrosis. The abscess formation, with localized peritonitis, which so often gives rise to symptoms which simulate appendicitis, save that they are present on the left side, is perhaps the most common complication of this disease. Rarely does a diverticulum perforate through into the abdominal cavity, causing generalized contamination. We have seen this occur, but usually the perforation is slow and walled off and results in local, rather than diffuse, peritonitis. The obstruction which results from diverticulitis is peculiar in that it is due to extrinsic inflammatory reaction and contraction of the underlying coats of the bowel, in contradistinction to that produced by ulcerating carcinoma in which the contraction begins from within and progresses outward. The slow inflammatory change that produces stenosis following abscess formation, or occasionally without it, usually results in a fistula, and in our experience these fistulæ have been extremely difficult of closure unless we resected the piece of bowel which contained the stricture. Closure without resection almost invariably fails.

The exact relation of carcinoma to diverticula is questionable. There is no reason why a carcinoma should not develop in the mucous membrane of the diverticulum and this does happen occasionally, but there is little evidence to support the view that carcinoma is the result of diverticulitis. Indeed, the association of the two lesions is so uncommon that one may question the diagnosis of carcinoma in association with diverticulitis, except under the most extraordinary circumstances. Occasionally carcinoma and diverticulitis are associated, and apparently one is able to demonstrate a carcinoma arising from a diverticulum or engrafted on a diverticulum, but the percentage of cases occurring thus is almost negligible. We have several times resected the sigmoid for diverticulitis, because there has been recent bleeding from the bowel, in the belief that carcinoma was engrafted on the inflammatory process. With rare exceptions, we have been unable to demonstrate such a change. The so-called mimicry of carcinoma, which diverticulitis has been accused of, is infinitely more theoretic than real. This seems the logical conclusion from a study of the 227 cases of diverticulitis in this series which actually required treatment; co-existing carcinoma was found in only 4 cases and during the course of operation for carcinoma of the bowel in 679 cases, diverticulosis was present in only 4. If, in such a large group, the conditions are found co-existent in only 8 cases, it seems reasonable to believe that the relationship is incidental rather than significant.

CLASSIFICATION OF DIVERTICULA

In order to evaluate more carefully the significance and importance of diverticula, we have used the following classification:

I. Diverticulosis, including that group of cases in which evidence of diverticula is found by roentgenographic examination or at necropsy and in which, from all available data, the diverticula do not bear any relationship to the patient's complaints.

II. Diverticulitis
  1. Acute
  2. Chronic
  3. Complicated
    a. Abscess formation
    b. Fistula
      External
      Internal { vesicocolic / enterocolic
      Multiple
    c. Associated with malignancy

Ordinarily, we consider acute, subacute, and chronic diverticulitis to be fundamentally medical problems and the complications essentially surgical. To serve as a basis for study, we have reviewed the cases of diverticula that occurred over a 5-year period, from 1923 to 1928. The patients in 48 cases of diverticulitis came to operation and 179 were treated by medical measures. After reviewing 254 cases in which diverticula occurred but

TABLE I.—INCIDENCE OF DIVERTICULUM ACCORDING TO AGE

		Decades						
	Cases	20–29	30–39	40–49	50–59	60–69	70–79	80–89
Clinical series	481	1	19	80	191	151	40	1
Necropsy series	111	1	...	6	30	41	31	3
Total	592	2	19	86	221	195	71	7
Per cent	...	0.03	3	15	37	32	10	1

seemed unrelated to any of the patient's complaints, we felt that nothing significant was being shown and discontinued further analysis of diverticulosis. Hence, this study includes the 481 cases mentioned, to which are appended figures from the necropsy service for this same period. In order to determine, if possible, the occurrence of diverticula, we secured information that there had been 24,620 roentgenograms of the colon, with a diagnosis of diverticula in 1,398 cases. This would suggest an occurrence of 5.67 per cent, but this figure is not entirely accurate as it is all but impossible to make deductions for the number of re-examinations that were made. However, in a figure this large, the error probably is not great and the figure may be of some significance because at necropsy of 1,925 cases (1924–1928), diverticula were found in 111 (5.2 per cent). It must not be held that 5 per cent represents the occurrence of diverticula for all ages, but refers more to a group of cases in which, for definite or even vague reasons, the colon was examined. The actual occurrence for all ages is probably less than 1 per cent, as estimated from the finding of diverticula in 2,310 cases, in the course of 765,795 examinations (1916–1928).

The situation of the diverticula is of interest. They occur most commonly in the sigmoid, but they may be distributed throughout the colon, usually with decreasing frequency from the left to the right side of the bowel. In the series found at necropsy, which is more accurate than clinical observation, diverticula occurred in the sigmoid in only 29 per cent; in the sigmoid and other parts of the colon, in 68 per cent; and in any part, but not in the sigmoid, in 3 per cent.

The question of the influence or significance of sex is small. In our series of 481 cases, 60 per cent were males, and in the series of 111 cases in which diverticula were found at necropsy, 70 per cent were males. The relation of age to diverticula is illustrated in Table I.

The fact that in the series of 111 cases in which diverticula were found at necropsy, all cases but one were in patients aged more than 40 years, as were 461 in the series of 481 cases, emphasizes the assumption that in patients aged more than 40 years the incidence of diverticula is about 5 per cent. It is not possible to estimate accurately the incidence of diverticulitis in relation to diverticulosis, but an approximate figure may be obtained from the 1,398 roentgenographic diagnoses in which are included 65 additional examinations of the colon of various patients with diverticula. Hence, in about 1,300 cases of diverticula, 227 are considered to be cases of diverticulitis, or approximately 17 per cent of the cases of diverticula seem to be productive of symptoms. This percentage, obtained from clinical observations, is somewhat higher than that obtained from necropsy, for in the 111 cases there were 16 cases of diverticulitis (14 per cent).

SYMPTOMS OF DIVERTICULA

Probably the most common symptom complained of is pain of some kind. Usually it ranges from an intermittent sharp pain, probably secondary to formation of gas, to a slow, boring type of discomfort which is present more or less constantly. There is no typical pain in diverticulitis, but the complaint is present in practically every case at some time during the disease. Usually it is situated in the lower left quadrant, or in the lower midabdominal section. Its reference depends largely on the accompanying complication, which usually is attachment to, or perforation of, another viscus. We have seen in the clinic 4 cases in this series in which the pain was referred to the right side, but this is extremely unusual. Constipation, as one would expect, is a rather constant accompaniment of diverticulitis, particularly when it has advanced to the complicated stage or when tumefaction, with encroachment on the lumen of the bowel, is present. In 142 cases (60 per cent of the series), constipation, either alone or alternating with diarrhœa, which was atypical, was

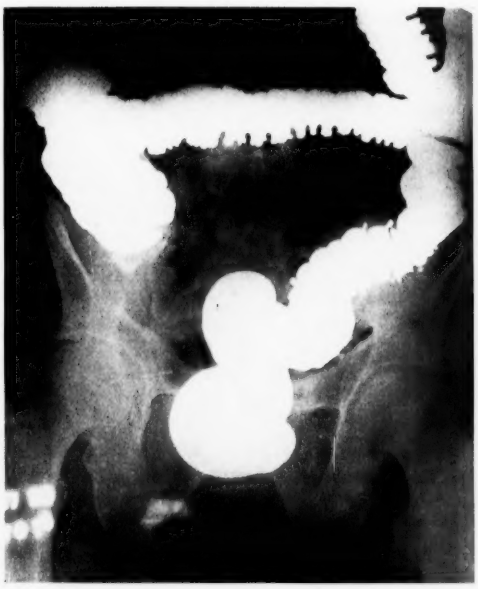

Fig. 1. Roentgenogram showing diverticula in the descending colon.

Fig. 2. Large diverticula of transverse and descending colon.

observed. On the other hand, diarrhœa alone was present in 35 cases (11 per cent), and although it was not true diarrhœa but usually more of a rectal tenesmus with the passage of a small amount of mucus, pus, and fæcal material, it gave sufficient disturbance of the intestinal habit to call the patient's attention to its presence. In 2 cases in this series, intestinal passage of apparently pure pus occurred, probably signifying the presence of an abscess which had ruptured into the lumen of the bowel in contradistinction to the usual course of penetrating the peritoneal coat.

Tumefaction was noted in 71 cases (31 per cent), and in a larger number a tender, easily palpable sigmoid was observed. Tumefaction, more often than not, represents merely a gross inflammatory reaction around segmental diverticulitis. Its mere presence is not a particularly serious circumstance and is not to be construed as indicating the more serious presence of a malignant condition, for in only 4 cases in which tumefaction was present was a malignant condition associated. On the other hand, unless the tumefaction is relieved by regression of the inflammatory process, obstruction, abscess, or a fistula is the result.

The presence of blood in the stool in diverticulitis is probably of small significance from a diagnostic standpoint, and although it was noted in 39 of the cases of this series, usually proctoscopic examination revealed the blood originating in the anal canal. In 20 of the cases, bleeding was demonstrated to be around the anal canal by proctoscopic examination, and, consequently, in the presence of tumefaction, a malignant condition was suspected, but, as has been noted, was found in only one of these cases. This is important, since previously we have been rather inclined to look on bleeding in the presence of tumefaction and diverticulitis as a rather constant sign of a malignant process and have urged exploration on this account. However, we believe that although one should be on one's guard for carcinoma in the presence of diverticulitis, particularly if blood in the stool is observed, although it is not a very reliable or trustworthy symptom.

Fig. 3. Marked spasm proximal to sigmoid with multiple diverticula distributed throughout the sigmoid and descending colon; diverticulitis of sigmoid.

Symptoms referable to the bladder are common and represent, in most instances, a rather serious complication: either attachment to the bladder or attachment to and perforation of the bladder. With a mobile sigmoid, which drops down into the extreme bottom of the pelvis, attachment to the bladder is easily accomplished by direct extension of the inflammatory process. Not only is it the most available viscus to become involved, but the situation of the involvement, which is usually at the lowest point on the bladder, also renders surgical interference, which is essential in many of these cases, excessively difficult and dangerous. In the surgical group of 48 cases in this series, urinary symptoms were definite in 13 (26 per cent), and in 7 of these there were fistulæ into the bladder with the accompanying passage of gas and fæces through the urethra. Although this is not always essentially a surgical condition, the danger of attending infection from the bladder renders any long standing vesico-intestinal fistula a serious condition. The diagnosis of this complication is readily established by cystoscopic examination, although one should always be able to suspect it from the knowledge of passage of gas or fæcal material, or both, through the urethra. In one case of this series, there was perforation into the ureter close to its juncture with the bladder and pyuria resulted, with a ureterovesico-intestinal fistula.

DIAGNOSIS OF DIVERTICULA

Laboratory aid in the diagnosis of diverticulosis consists chiefly in roentgenological examination (Figs. 1 to 5). Since the accurate diagnosis is made only by demonstration of the diverticula, and since their involvement in an inflammatory process gives characteristic roentgenological signs, the roentgenological examination holds first place among diagnostic procedures in the establishment of the diagnosis. In The Mayo Clinic the barium enema, observed roentgenoscopically, is used exclusively in these cases; the barium meal is eliminated in an effort, of course, to prevent further intestinal stasis by the introduction of a large amount of barium proximal to the suspected lesion. Diverticula manifest themselves roentgenoscopically and roentgenographically as rounded, knob-like projections from the lumen of the colon and show considerable variation in size. The sigmoid segment is the favorite site, and the diverticula become less numerous as the examination proceeds more proximally.

Roentgenological evidence of the presence of diverticulitis consists principally of the signs of extreme irritability that always are present with inflammation of a hollow viscus. These signs are spasm and hypermotility, and they vary in intensity with the extent, severity, and virulence of the process. All degrees of spasm are seen, from the mild type manifested by a sharp, serrated appearance of the haustra in a somewhat narrowed segment of bowel to almost complete occlusion of the lumen. The filling defect almost universally encountered is either of one type or a combination of two types: a false filling defect resulting from spastic narrowing of the affected segment which may be so marked as to produce complete occlusion, or a true filling defect resulting from encroachment of pericolic

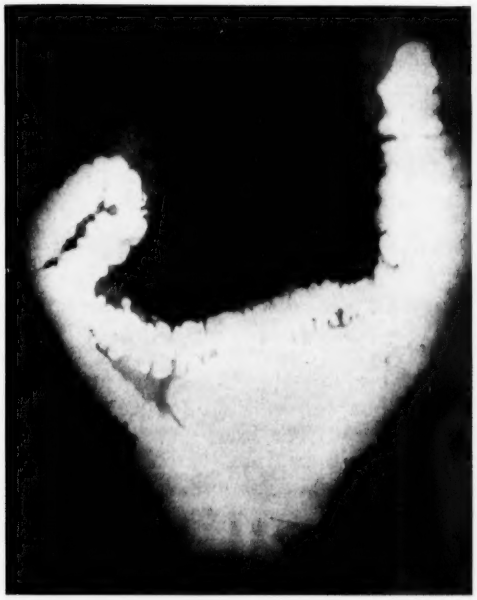

Fig. 4. In this patient the diverticulosis involved the entire colon.

Fig. 5. Diverticula in cæcum, descending colon, and sigmoid.

inflammatory tissue on the lumen of the bowel. Antispasmodic drugs, administered until the physiological effect is obtained, will modify the appearance at least of the former but will have little effect on the latter except to relieve concomitant spasm. These filling defects make the roentgenological differential diagnosis of diverticulitis and carcinoma confusing, but it can usually be accomplished by careful and painstaking observation. Differential points are the somewhat concentric contours of the segment noted in diverticulitis, contrasted with the sharply irregular contours in carcinoma, the maintenance of mobility in the former, compared with the stark immobility of the latter, and the relatively long segment of colon involved with diverticulitis, whereas carcinoma usually involves a much shorter segment.

Proctoscopic examination is of relatively little value in the diagnosis of diverticulitis, save when the lesion is extremely low and the distal portion may be visualized. In the medical group of 179 cases, proctoscopic examination was made in 83. Reports were as follows: 60, negative for abnormality above the anus; 14, immobile or sacculated sigmoid; 4, pelvic mass; and 5, sufficient visualization to allow of a diagnosis of diverticulitis. Proctoscopic examination was made in 36 of the 48 surgical cases, with negative results in 9, immobile sigmoid was reported in 3, sigmoidal or pelvic mass in 9, and diverticulitis in 10.

The blood picture in diverticulitis is of some diagnostic value but is not of great significance. In 11 of the 48 surgical cases, the hæmoglobin was less than 70 per cent, and of these there was carcinoma in 2 cases and an associated bleeding duodenal ulcer in 1 case. Anæmia is an uncommon accompaniment of diverticulitis and, when present, indicates usually either a long standing infection or possibly an associated malignant condition.

There is no absolute type of persons more prone to the development of diverticulitis than others, but there is a distinct tendency in a certain group of persons who conform to a common anatomical type: a middle aged man, preferably a physician, inclining toward corpulency and leading a sedentary existence,

Fig. 6. Carcinoma of the colon developing in the presence of a diverticulum.

in whom, with increasing years and a tendency to constipation, a syndrome develops of left sided lower abdominal irritation, possibly pain, and other symptoms associated with advancing inflammatory reaction. This type, more often than any other, inclines toward diverticulitis.

MEDICAL TREATMENT

The treatment of diverticulitis is preferably medical and usually only when complications occur is operation to be undertaken. The presence of a tumor, especially if associated with obstruction, arouses fear that the trouble is malignant and if the other clinical data, particularly the history, do not tend to support the diagnosis of diverticulitis, operation must be carefully considered. Medical treatment in acute cases consists essentially of rest in bed, residue-free diet at the onset, icebags to the lower part of the abdomen, and rectal irrigations with hot physiological solution of sodium chloride. As the condition subsides in the course of a few days, a bland, anticonstipation diet is instituted and mineral oil is given orally. In the use of the mineral oil, we believe it preferable to administer only 4 to 8 cubic centimeters three times daily, rather than 15 to 30 cubic centimeters once or twice daily. Excessive oil merely leaks through the rectum in many instances and gives rise to the desire to discontinue its use. Used in small doses, this objection seldom arises, and we consider the constant lubrication of the area of the diverticula of such importance as to necessitate continuation of the oil indefinitely. The hot irrigations are discontinued as soon as the inflammatory reaction subsides and the bowel begins to empty naturally. We are not sure of the value of tincture of belladonna but since it may help to relax the intestinal spasm, it is administered in doses of a 0.33 to 1 cubic centimeter three times daily.

On the patient's dismissal, constant diligence in the care of the bowels and daily use of mineral oil must be emphasized. Even in cases of diverticulosis, this advice is indicated, since it may minimize the potential danger of diverticulitis.

Data on the results of medical treatment are meager; of 37 patients who were treated medically, only 2 later came to operation. One had gone along without incident for 6 months, only to suffer a recurrence which rapidly resulted in the establishment of a vesical fistula. The second patient had been under observation for 5 years and had been at the clinic several times for treatment of exacerbation of the diverticulitis. Each time the disease subsided, but the patient failed to carry out anticonstipation measures at home. The patient, wearying of trying to get along, elected operation. Two other patients, of the 37, had fairly severe recurrences but they were controlled by medical measures. The remaining 34 have been free, or practically free, of symptoms for 6 months to 7 years. It was not uncommon to have reference made to some pain and distress, if the bowels were not

TABLE II.—TYPES OF OPERATION AND CAUSES OF DEATH IN HOSPITAL

Type of operation	Cases	Cause of death	Cases
Mikulicz	10	Shock	1
Colostomy	8	Obstruction from diverticulitis and acute ulcerative colitis	1
Cæcostomy	1	Pulmonary embolism	1
Colostomy and resection or drainage	22	General peritonitis and gangrenous cellulitis of abdominal wall	1
Repair of vesico-intestinal fistula	2	Pulmonary suppuration with effusion	1
Exploration	2	Postoperative hæmorrhage	1
Exploration and separation of adhesions and closure of fistulas of the small bowel	1		
Plastic on sigmoid and appendectomy	1		
Cholecystectomy	1		

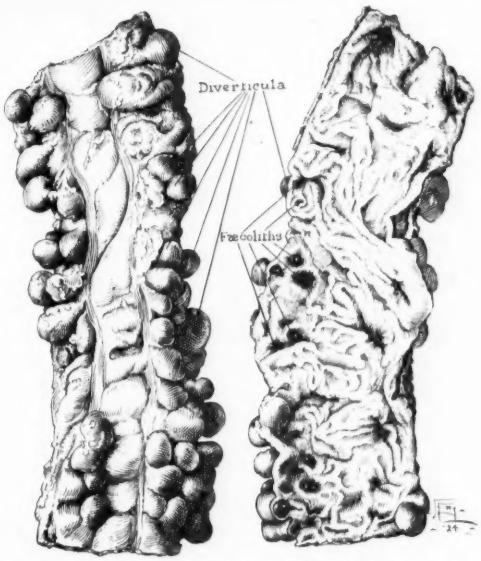

Fig. 7. Multiple diverticula of the descending colon filled with fæces.

carefully regulated. It is probable that some of the patients in the large remaining group concerning which later data are not available, have suffered further trouble or have even suffered from complications. Yet, we believe that most of them will get along satisfactorily if they persist in the care of the bowels and the use of mineral oil.

SURGICAL TREATMENT

Surgical interference, because of the mere presence of diverticula, or even in the early inflammatory stages of the disease, is perhaps not usually indicated and frequently is unwarranted. We believe it more essential to confine surgical operation, in this ailment, to chronic complicated cases or to cases of the acute type in which the condition has progressed to perforation. The complications which arise and necessitate surgical intervention are: (1) acute perforation; (2) abscess; (3) fistula, whether external, vesical, intestinal or multiple; (4) inflammatory obstruction; and (5) malignancy (Figs. 6 and 7).

Fortunately, acute perforation of a mobile segment of the colon, where diverticulitis more frequently occurs, is unusual. We have seen it in an occasional case in the clinic but it is less common than perforation into the free peritoneal cavity from carcinoma of the colon. Usually, perforation from diverticulitis is not into the free peritoneal cavity because the inflammatory reaction most commonly draws to the sigmoid either loops of the small bowel or fixes the sigmoid to the lateral parietal peritoneum, bladder, or anterior abdominal wall. Consequently, penetration and abscess more commonly result. In acute perforation, the ideal type of procedure is to remove the offending diverticulum, close the opening, and drain the peritoneal cavity. Our experience is small in this type of case and the mortality rate is high. Abscess, however, is not an infrequent occurrence and demands surgical intervention. Abscess may form against the anterior or lateral parietes and may perforate through the abdominal wall, as we have seen it do in one case, or it may perforate into a viscus. When it perforates through the abdominal wall, a serious condition confronts both patient and surgeon. This usually is the result of long standing inflammation and is accompanied by obstruction from stricture.

We have found, in a small proportion of these cases, that the most usual operation demanded (Table II) has been removal of the affected sigmoid, with end-to-end anastomosis, following drainage by colostomy farther

back in the colon, preferably in the transverse colon. Plastic operations usually are of no avail in this complication, and in the presence of acute or subacute inflammation certainly one should not undertake such a formidable procedure as resection. Consequently, it is usually wiser, we believe, to perform a drainage operation and allow the patient to return home for 2 to 4 months, applying local treatment to the inflammatory area through the rectum and the colostomy opening. Often the recession is so marked that subsequent removal of the offending segment may be accomplished with little danger. Likewise, in the cases of rather acute, diffuse diverticulitis, with tumefaction in which one feels that resection should be done because of the extent of the disease and the obstruction present, we have found it most satisfactory to perform a drainage operation and to postpone removal of the lesion for a considerable length of time. In 1 or 2 cases, we have been favorably impressed by the great recession of the growth that has taken place after drainage. In fact, we believe that frequently, after colostomy has been done in a case of rather diffuse diverticulitis, and after the consequent "sidetracking" has been carried on over a sufficiently long period, the recession will be sufficient to allow of omission of subsequent resection. Obviously, one must be sure, before closing the colostomy opening and abandoning the idea of further operation, that there is no obstruction at the primary site of the disease.

Formation of fistula leading into the bowel or the bladder is a serious complication, particularly fistula leading into the bladder. Here the inaccessibility of the two openings makes the surgical procedure extremely difficult. Formerly, we were inclined to attempt this type of procedure in one stage, closing the two openings and hoping for primary union. There is always a certain amount of infection around the field and thus a graded operation, namely, colostomy first and subsequent attention to the fistula, may be done with lower mortality and more satisfactory end-results.

When carcinoma is believed to be present, even though one may not be absolutely sure of it, resection is indicated. In any case of diverticulitis in which the diverticulum is suspected of harboring a malignant process the diverticulum should be removed. The type of removal depends on the opinion of the surgeon in many cases, but in our experience a radical operation has been indicated in only a few cases in the presence of inflammation of any extent. The operation of choice is colostomy and subsequent removal of the growth after regression of the inflammatory reaction.

SUMMARY AND CONCLUSIONS

1. Diverticulosis is quite prevalent, apparently occurring in about 5 per cent of persons who have symptoms referable to the large bowel, but probably actually occurring in about 1 per cent of all persons.

2. Diverticulitis probably occurs in about 17 per cent of cases of diverticulosis and in most instances is chronic in its course and subject to exacerbations.

3. The etiology of diverticula is obscure, but they are probably the result of several factors, among them inherent muscular weakness in the wall of the bowel and environmental conditions, obesity, and constipation.

4. Diverticulitis probably is the result of improper emptying of the bottle shaped sacculations, with subsequent inflammatory reaction, necrosis, and occasional perforation.

5. The relationship of diverticulitis to carcinoma probably is incidental rather than actual.

6. In 227 cases reviewed in this paper, as treated at The Mayo Clinic, a malignant condition was found associated in four only.

7. Diverticulitis occurs almost entirely in persons of middle age who are inclined to be corpulent and who lead sedentary lives. Diverticulitis usually runs a chronic course with several exacerbations and yields satisfactorily to dietary and medical treatment.

8. The outstanding symptom of diverticulitis is pain, usually situated in the lower left portion of the abdomen and is frequently associated with constipation. Change in bowel habit is a confusing factor.

9. Bleeding is not commonly found among the symptoms of uncomplicated diverticulitis or diverticulosis. When it does occur, an associated malignant condition is always suspected but frequently not found.

10. Tumefaction associated with diverticulitis is common and usually is the result of inflammatory reactions, with or without formation of the abscess. In itself, it does not indicate associated malignancy.

11. The medical treatment of acute diverticulitis consists of watchful waiting while the patient is at rest in bed and is given irrigation of the affected segment of bowel with warm sodium chloride solution and other sedative solutions. As the process subsides, anticonstipation diet and the use of small doses of mineral oil orally are given. A dietary regimen is highly essential and probably often prevents complications.

12. In a definite percentage of cases, diverticulitis tends to become complicated. The most common complications are abscess, fistula, and perforation.

13. The treatment of the complications of diverticulitis is usually surgical, particularly of the internal fistulous formation in which a viscus, such as the bladder, is penetrated by the inflammatory process.

14. Primary resection in the face of complications and diffuse inflammation is accompanied by a relatively high mortality rate. The operation of choice is a graded procedure, consisting of drainage and subsequent resection and anastomosis.

15. Often prolonged drainage by colostomy permits complete recession of tumefactions and disappearance of clinical symptoms, rendering unnecessary further intervention.

MALIGNANT TUMORS OF THE NAIL BED

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IN malignant tumors the early stages are often masked by a harmless appearance and insignificant symptoms so that they may be overlooked and the best chances for cure are lost. Those afflicted with benign tumors, in the course of years become more less accustomed to them so that the transformation to a malignant stage is often overlooked until the new-growth has advanced so far that there is little possibility for a successful removal. Beginning malignant tumors not seldom resemble inflammatory conditions and are mistaken for such, especially if they occur in places frequently exposed to slight injuries and, therefore, places in which infections and inflammations are common. This is well illustrated by a pigmented tumor which arises at the border of, or beneath the nail. Located between nail and bone with little soft tissue to expand in the tumor destroys the nail and often is still of no considerable size even when the regionary lymph glands or distant organs have become invaded by metastases. Since pigmentation may be slight and secondary infections may change the appearance of the exposed surface, the easily bleeding, granular mass breaking through the nail suggests an innocent granulation tissue until microscopic examination reveals the true nature of the growth.

From a review of the literature it seems that the melanotic tumors of the nail bed are rare. Since the first description by Boyer in 1854 and Demargnay and Monod in 1855 only about 27 cases have been reported (Womack). Hutchinson has called the condition "melanotic whitlow" which indicates both the resemblance to an inflammatory lesion and the pigmentation. The majority of these tumors were found on the fingers, especially on the thumb (13 cases) while on the toes only 4 have been observed, namely 3 on the great toe (Jones 2 cases, Bonnet) and one on the little toe (Chauvenet and Dubreuilh). These tumors are very malignant and usually come under observation after metastases have developed in the regionary lymph glands or in the internal organs.

I recently had the opportunity of examining a characteristic subungual melanoblastoma of the great toe and since so little is known about this tumor and it may so easily

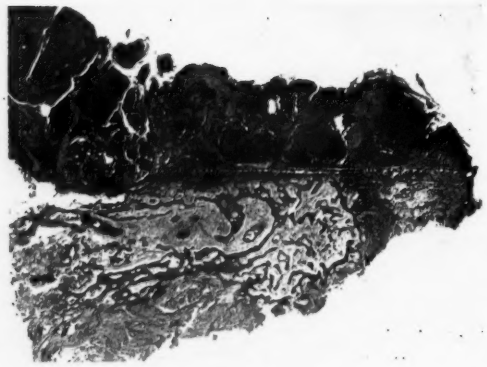

Fig. 1. Subungual melanoblastoma of the great toe. The nail bed is the site of a cellular tumor the surface of which is ulcerated. The tumor extends close to the bone which shows rarefication of the trabeculæ and fibrosis of the bone marrow.

be overlooked, a description of it seems to be warranted.

SUBUNGUAL MELANOBLASTOMA OF THE GREAT TOE

A white German woman, aged 69 years, complained of pain in the right great toe which had been noticed for 3 years. She attributed the pains first to a poorly fitting shoe. During the last year the toe became swollen and bled on several occasions. A physician who saw her about a year ago diagnosed an inflammation of the nail bed, made an incision, and prescribed wet dressings. Since the wound did not heal but grew larger, the patient went to the hospital for further treatment.

The examination revealed a well nourished woman whose past history is negative except for a rupture of the gall bladder several years ago. According to her knowledge there were no cases of carcinoma in her family. She had 3 children who are living and well. She complained of dyspnœa, shortness of breath, precardial pain, and palpitation. The heart was slightly enlarged to the left. Blood pressure was 178 –100; pulse rate, 92. The urine showed a trace of albumin, a few pus cells, and an occasional hyaline cast. Blood count showed red cells, 4,800,000; hæmoglobin, 75 per cent; white cells, 7,400; leucocytes, 62; lymphocytes, 31; monocytes, 5; and eosinophiles, 2 per cent.

The nail of the right great toe was almost completely replaced by a soft, dark red, and easily bleeding mass. The terminal phalanx was moderately swollen and slightly tender to touch. X-ray examination of the bone was negative. In the right groin there was a mass of enlarged lymph glands, the largest having the size of a hen's egg. Under local anæsthesia the terminal phalanx was removed. The patient had an uneventful recovery and left the hospital after 8 days. The enlarged glands in the groin were treated with deep X-ray therapy and decreased in size. For the last 6 months they have remained stationary.

The specimen consisted of the amputated terminal phalanx of the right great toe. The nail bed was transformed into a roughly oval ulcer, 24:27 millimeters in diameter. The edges were sharp, slightly indented, and in places undermined for 1 to 2 millimeters. The floor of the ulcer was firm, finely granular, of light purplish gray color, and was covered with a thin, adherent, light yellow gray membrane. Near the posterior border there was an irregular deep brown line. Of the nail only a small fragment was left. It occupied the posterior medial part of the nail bed and measured 20:5 millimeters in diameter. A longitudinal section through the middle of toe showed the purplish gray tissue of the ulcer extending close to the bone and measuring 10 millimeters in vertical diameter. Its posterior part contained several deep brown areas up to 5 millimeters in diameter.

Microscopic examination. The nail bed was the site of a very cellular tissue extending close to the terminal phalanx from which it was separated by a thin layer of fibrillar connective tissue. Branched septa of connective tissue divided this tissue into spherical areas from 1 to 2 millimeters in diameter (Fig. 1). From the septa delicate fibers and capillary blood vessels extended into the cellular areas and subdivided them either into small irregular alveoli or narrow cords.

The cells of which the tissue was composed were large and of varying shape. They were round or oval or polygonal and by compression often assumed a spindle shape. These spindle shaped cells had the tendency to fascicular arrangement.

The cells possessed an ample clear cytoplasm and large round or oval nuclei. The largest of the cells contained two nuclei. The chromatin of the nuclei was finely granular and was evenly distributed. The nuclear membrane was distinct and there was either a small basophilic or a large oxyphilic nucleolus. There were from 3 to 5 mitotic figures to high power field.

The brownish discoloration in the posterior part of the tumor was due to the intracellular accumulation of dark brown pigment granules. The cells filled by the pigment were elongated and branched and the pigment granules which were of even size extended into their branches. In the clear round, oval, and polyhydral cells no pigment could be demonstrated. Pigmented and non-pigmented cells often mingled with each other (Fig. 2).

The septa contained an occasional accumulation of lymphocytes and single pigmented cells. In these cells the pigment granules were lighter and of irregular size (phagocyted melanin). In none of the locations did the pigment give the iron reaction. It was slowly bleached by hydrogen peroxide.

There were regressive changes in the center of the larger alveoli as well as on the free surface of the

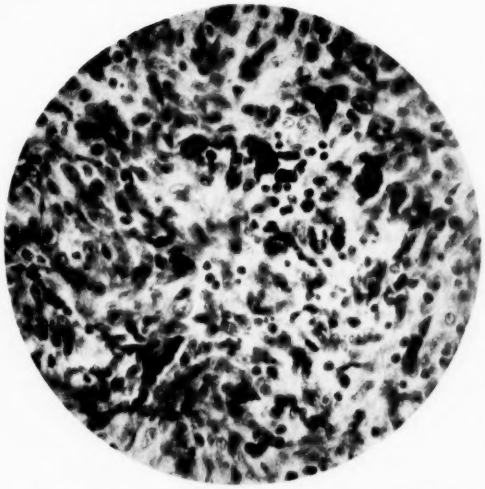

Fig. 2. High power magnification of an area near the posterior border. Note the large clear cell free from pigment and the elongated and flattened elements filled with deep brown pigment granules.

tumor. The latter was covered with a layer of fibrin, necrotic cells, and degenerated pus cells. In places the tumor tissue bordered directly on stratified epithelium.

The bone was not invaded by the tumor. It appeared rarefied with thin and scanty bony trabeculæ and an ample marrow composed of fibrillar connective tissue with perivascular accumulations of plasma cells and lymphocytes (Fig. 1).

Histological diagnosis: melanoblastoma of the nail bed. Rarefying osteitis of the terminal phalanx.

The papers dealing with the melanotic tumors of the nail bed say little about the differential diagnosis. There are several benign tumors which occur in this location and which will be discussed later. Occasionally a squamous cell carcinoma may arise in the region of the nail. Its clinical picture resembles that of the melanoblastoma as illustrated by the following case.

SQUAMOUS CELL CARCINOMA OF THE NAIL BED OF THE LITTLE TOE

A Russian Jew, 63 years of age, complained of pains in the little toe of the right foot which had been bothering him for several years. These pains had been present all the time but had become more severe during the past few months. The terminal part of this toe was found transformed into a dry, firm mass and the clinical diagnosis of senile gangrene of the little toe was made. Under local anæsthesia, the middle and terminal phalanges were removed. The wound healed *per primam*, and the patient left the hospital after 5 days. There were no enlarged glands in the groin or elsewhere.

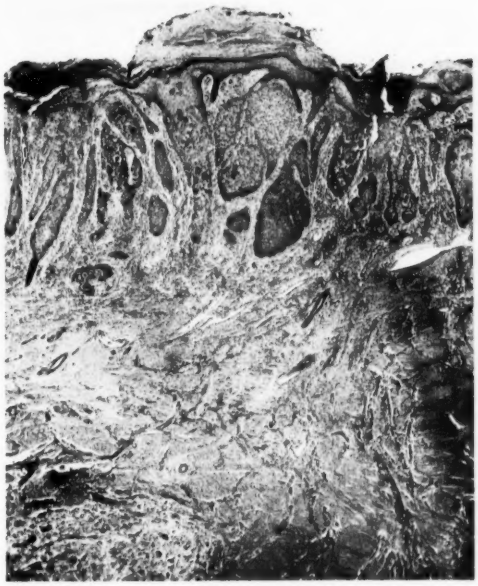

Fig. 3. Subungual squamous cell carcinoma of the little toe. Note on the surface the remnant of the nail and the proliferation of the rete malpighii. ×24.

The specimen consisted of the little toe of the right foot enucleated in the joint between middle and basal phalanx. The nail was replaced by an irregular ulcer which extended over the upper part of the anterior aspect of the toe. The ulcer measured 25 : 12 millimeters in diameter and had slightly raised and scalloped edges. The floor was firm, dry, scaling, and of waxy appearance. There were a few pinhead sized depressed areas which were purplish gray in color.

Microscopic examination. The dry and waxy tissue on the surface of the ulcer was revealed to be composed of a thick layer of hornified material which was invaded by degenerated pus cells. This material covered irregular, branched, and budding papillæ which extended deep into the cutis and were surrounded by dense accumulations of lymphocytes and plasma cells (Fig. 3). The papillæ were composed of cells, some of which had still retained their prickle shape while others were polyhydral with large and indented nuclei. There were many atypical mitotic figures. In the center of some of the papillæ concentrical rings of hornified material were present. The bone appeared unchanged.

Histological diagnosis: squamous cell carcinoma of the nail bed.

The malignant tumors of the nail bed can be easily distinguished from the benign tumors because the latter do not break through the nail. These benign tumors are: the subungual fibroma (Leduc and Suter), the Dupuytren's subungual exostosis, and a peculiar new-growth the origin and nature of which has been much discussed. This tumor has been described under different names such as angiosarcoma, colloid sarcoma, and perithelioma, and though its microscopic appearance is somewhat suggestive of a sarcoma the clinical course is benign. According to Masson, the tumor originates in the neuromyoarterial glomus which is arranged about the small arteries of the skin and is composed of smooth muscle fibers and nerve cells. The glomus tumors are non-pigmented and consist of blood vessels, muscle fibers, nerve cells, and nerve fibers. They are most frequently found beneath the nail (Masson, Martin and Dechaune, and Nicod) but occur also in other places of the extremities (Masson and Gery, Psodanoff). The subungual glomus tumor appears as a blue spot or nodule which is very painful. The pains are excruciating and radiating. The tumor does not destroy the nail, although it may produce a slight depression in the bone. Metastases have never been observed.

In order to distinguish the melanoblastoma from harmless granulomata, the demonstration of pigmented areas or of a pigmented line near the border is of great significance. This pigmentation, though often very slight and visible only with the aid of a magnifying glass, secures also the differentiation from a squamous cell carcinoma of the nail, the surface of which is dry and waxy.

The malignant tumors of the nail bed occur in higher age. No case of "melanotic whitlow" has been reported in a patient under 35 years of age and most of the patients are in the sixties or seventies. In about half of the cases of melanoblastoma, a trauma is reported to have preceded the tumor for from 1½ to 5 years. In 3 cases the melanoblastoma seems to have started from a subungual pigmented mole.

Especially as far as the melanoblastoma is concerned, the prognosis is grave. Absence of local metastases does not exclude the involvement of internal organs (Chauvenet and Dubreuilh, Womack, 3 cases). On the other hand, patients with enlarged regionary lymph glands may survive the amputation of the toe or finger for several years. In the cases in which the lymph glands were not submitted for microscopic examination, it remained doubtful whether the enlargement was due to a metastatic invasion or to a chronic inflammatory irritation.

The treatment of the melanoblastoma of the nail bed is the early radical operation. Most authors agree that X-ray or radium are contra-indicated. The average length of life after the diagnosis is made is 14 months.

CONCLUSIONS

Ulcerative lesions of the nail bed in elderly persons which do not show any tendency to heal should be examined carefully for a possible melanoblastoma or squamous cell carcinoma. In distinguishing the melanoblastoma from benign granulomata and squamous cell carcinoma, the demonstration of pigmented areas which may be very small is of great importance. In this location the carcinoma shows a dry and waxy surface.

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THE ABSORPTION AND TRANSFERENCE OF PARTICULATE MATERIAL BY THE GREAT OMENTUM[1]

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FUNCTIONS of the omentum, such as the elaboration of peritoneal fluid, the absorption of particulate materials or the production of antibodies, have long been known. Ever since Aristotle attributed to the great omentum a function to protect abdominal viscera from the cold, each succeeding scientific generation has contributed its experimental data to our knowledge of the part the organ plays in regulating body economy. Although the literature on the anatomical, physiological, clinical, and surgical significance of the omentum is enormous, complete and exact data concerning it must yet be compiled.

The removal of isotonic solutions and numerous suspensions of particulate material from the peritoneal cavity has been a favorite subject for study since von Recklinghausen described stomata within the mesothelium of the cœlom through which such particles may be removed. Any study of the method of removing foreign particles from the peritoneum, the type of cellular response to invading organisms and the routes of drainage from the peritoneum, must of necessity include the reaction manifested by the greater omentum.

Whenever foreign particles, such as India ink, trypan blue, or whole blood, are introduced into the peritoneal cavity, a polymorphonuclear leucocytic reaction follows within the first few hours; mononucleated cells appear in abundance only after 24 to 48 hours. McJunkin (1925) recognized three types of cells determined by the manner in which they take up neutral red in supravital staining. Whether these cells, in large part, are derived from the omentum has not been determined.

The large number of histocytes which abound in the normal omentum leads one to the opinion that this organ may be the source of many of the peritoneal exudate cells and yet experimental evidence does not thus far warrant such a conclusion.

The literature reporting the cellular response within the peritoneal cavity to any irritation is enormous, and we shall not attempt to review it here. Generally, these exudate cells which arise following peritoneal injection have been attributed to the mesothelium lining the cavity, the reticulo-endothelium of the spleen and lymph nodes, the specialized vascular endothelium of the liver, and the greater omentum.

Using a new finely particulate graphite suspension, Higgins and Graham (1929) undertook a study of the rôle of the diaphragm in the removal of foreign particles from the peritoneal cavity in the dog. Special emphasis was placed on the lymphatics of the diaphragm and the routes of absorption through the anterior mediastinum. Higgins, Beaver, and Lemon (1929) continued the study of absorption through the diaphragm of dogs which had been previously subjected either to unilateral or to bilateral phrenic neurectomy. Diaphragmatic paralysis, which follows section of the phrenic nerve, merely retards the rate of absorption, but it does not render the diaphragm any the less effective for the removal of injected material from the peritoneum.

During this study of absorption through the diaphragm, we were impressed by the speed with which the great omentum in these animals, absorbed and fixed the graphite particles. It was apparent at once that the mesothelium covering the omentum reacted far more intensively than the other surfaces of the peritoneum. Poynter (1928) stated that the peritoneum covering the omentum is different from that which covers the visceral and parietal surfaces. Furthermore, we have noted, in our study of the relation of the omentum to diaphragmatic absorption, that

[1]Submitted for publication October 23, 1929.

animals from which the omentum had been partially resected were not able to withstand the effect of graphite within the peritoneum and usually died within 2 to 3 weeks following an injection. In experiments on dogs that had been subjected to partial resection of the omentum some time before peritoneal injection, graphite particles appeared on the pleural surface of the diaphragm following injection as rapidly as in the intact animal. Rubin (1911), however, in studying absorption of indigocarmine from the peritoneal cavity of cats from which the omentum had been resected previously noted marked delay in the appearance of the dye in the urine from that which takes place in a normal animal. From our studies it seemed evident that the great omentum was essential to adequate protection, but that it did not bear any relation to the absorption of particulate material through the diaphragm.

Restoration of an organ is rather positive evidence that it is essential for the well-being of the organism. Its restorative capacity is great and surgeons have noted that at a second laparotomy following earlier partial resection of the omentum it has been restored to its earlier proportions. Arnaud (1928), in the most recent work on the omentum, stated that in both guinea pigs and dogs complete regeneration following resection has taken place in 5 to 6 weeks.

In our study on the absorption of graphite from the intact peritoneum, the abundance of black particles within the omentum and the gross appearance suggesting a system of drainage from the organ to the gastric and hepatic regions naturally prompted the inquiry as to whether the omentum could remove absorbed materials and, if so, whether such drainage passes by way of the diaphragm to the thorax. We were interested to know whether material engulfed by the omentum was removed through definite channels, carried by way of the blood stream, or perhaps remained as isolated foreign substances within the organ itself.

It is difficult to study the absorptive mechanism of the omentum, apart from the other closely related structures of the peritoneal cavity, because the omentum must be withdrawn from the cavity entirely or be isolated in such a way as to be independent of the adjacent organs. Shipley and Cunningham (1916) were perhaps the first to make a histological approach to the study of omental absorption, and they overcame the spatial difficulty by withdrawing the omentum from the cavity of the body and immersing it in a fluid or suspension, the absorption of which they wished to study. In this way all other drainage routes from the peritoneal cavity were necessarily excluded. In order to maintain anæsthesia over periods sufficiently long to permit adequate absorption, decerebrate animals were used so that mechanical difficulties were largely overcome. In this way the omentum could be easily immersed in various solutions, and suspensions and studies were made on the rate and the routes of absorption by the omentum in animals so prepared. In certain of the animals, Shipley and Cunningham previously had ligated the thoracic duct in the neck, attempting thereby to determine whether or not the lymphatic system was concerned in absorption by the omentum. Their results were unchanged by the ligation of the thoracic duct. In this connection, however, it must be recalled that should drainage from the omentum pass by way of the diaphragm into the anterior mediastinum a large portion of the lymph will enter the blood stream by way of the right cervical duct so that ligation of the thoracic duct would not be a completely effective control. They concluded that absorption of both solutions and suspensions is by way of the omental veins and that granular material may be recovered from the portal vein and liver within a very short time following immersion in the solution. They stated that lymph vessels, if present within the omentum, do not play a part in the removal of particulate material. Likewise Poynter (1928) in studying the functions of the great omentum, is convinced that the removal of foreign particles is accomplished by the omental and the portal veins. Poynter did not mention a lymphatic system, but he demonstrated crystalloid, and various particles and granules in the portal vein within a few minutes following peritoneal injection.

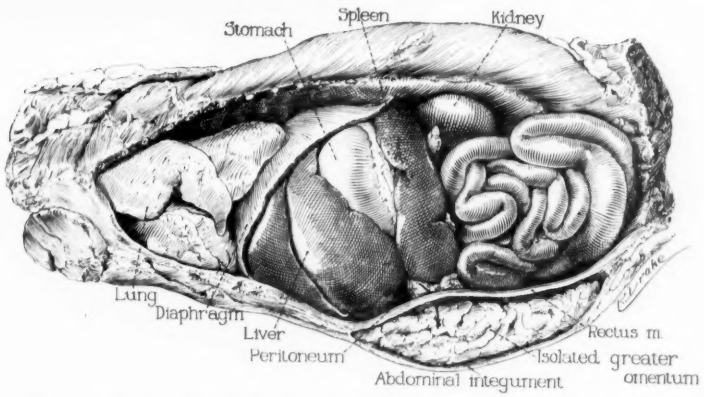

Fig. 1. Side view of the body of a cat, showing the position of the subcutaneous pouch, the isolated portion of the omentum contained therein, and the relation of the abdominal viscera to the isolated omentum.

In our earlier study on peritoneal absorption, we were unable to recover graphite particles from the liver for a considerable period following peritoneal injection. Furthermore, in sections of the omentum taken at various times following peritoneal injection, fixed in Zenker and formol solution and stained with hæmatoxylin, and with eosin-azure, we were unable to recognize any graphite particles in any of the omental blood vessels. Numerous particles were adherent to the surface mesothelium and a considerable portion of free graphite was present within the layers, but most striking perhaps was the exceedingly large number of histocytes packed with the phagocytosed particles.

These earlier observations prompted us to try a series of experiments in which we could isolate the omentum from the peritoneal cavity and thus study absorption as carried on by the omentum alone, apart from other adjacent organs and under conditions approaching the physiological. We hoped, too, to determine whether the lymphatics of the diaphragm and the anterior mediastinum had a part in drainage from the omentum as well as in drainage directly from the peritoneal space.

EXPERIMENTAL METHOD

Cats were used in this study. In order to avoid the necessity of decerebration and exposure of the omentum to atmospheric conditions, we isolated the distal two-thirds of the omentum in a pouch formed within the ventral abdominal wall (Fig. 1). With the use of Narath's modification of the Talma-Morison operation, under ether anæsthesia and sterile technique an incision about 4 centimeters long was made in the abdominal wall slightly to the right of the median line. By dissecting away the intervening fascia, a pouch of considerable proportions was made between the integument and the layers of muscle. The muscle tissue was incised in the median line, a small incision was made through the peritoneum, and the great omentum was carefully withdrawn through this incision and gently placed in the pouch previously prepared between the integument and the abdominal muscles. Caution was observed to avoid any undue manipulation, such as tension on the stomach, spleen, and the transverse colon. In the cat, the omentum is extensive and in this way a considerable portion (approximately two-thirds) may be withdrawn and thus isolated from the peritoneal cavity. Sutures were inserted around the opening in the peritoneum, fixing it firmly to the omentum, so that there might be no spatial continuity between the pouch beneath the integument and the peritoneal space. The skin was then closed. This gave an operative hernia of the omentum. The animals speedily recovered from the operation and in 4 to 5 days, ample

Fig. 2. Portion of the omentum within the subcutaneous pouch 3 hours after an injection of graphite.

time for the wound to heal, 1 to 5 cubic centimeters of the graphite suspension was injected into the pouch containing the isolated portion of the great omentum. Care was exercised in this injection, the needle being introduced through the integument into the posterior region of the pouch to avoid actually injecting the omentum. Following these injections, animals were killed at successive intervals; the isolated portion of omentum, the peritoneal portion of omentum, the gastrohepatic omentum, the diaphragm, the anterior mediastinum, and the liver were examined for evidence of absorption of the graphite. Fourteen experiments were considered successful. Careful necropsy was performed at intervals following injection, ranging from 30 minutes to 48 hours. These form the basis for the conclusions presented in this report.

EXPERIMENTAL OBSERVATIONS

One of the functions of the great omentum is the elaboration of fluid with marked coagulative properties. Arnaud (1928) recently demonstrated the secretory activity of the omentum in guinea pigs by inserting into the abdominal wall a window of transparent cellophane so that he could observe the movements and responses of the omentum. He concluded that the omentum is the source of large quantities of a serous fluid which possesses marked coagulative properties. Our observations bear out these conclusions in that foreign particles in contact with the omentum become at once adherent, held by a coagulum to its mesothelial surface. Within a few minutes following an injection of graphite into the abdominal pouch, the omentum is well speckled with particles which may be washed off only with considerable difficulty. The proximal portion of the omentum within the peritoneal cavity is already faintly gray in places, suggesting graphite, and sections show that the black particles have extended along the mesothelial surface and are still largely adherent. A few very fine granules lie beneath the surface layer. Thus far a phagocytic reaction has not been marked, for only occasionally we encountered phagocytic cells just beneath the mesothelial surface containing only a few black particles.

In a cat killed 3 hours after an injection of 3 cubic centimeters of the graphite suspension into the subcutaneous pouch, absorption by the omentum was well advanced (Fig. 2). The fluid then within the pouch contained many polymorphonuclear leucocytes with black granules, but whether these arose from the omentum or came from other sources as a result of irritation is as yet unknown. Diapedesis through the mesothelium into the surrounding area was not extensive, and we are inclined to believe that more stress is placed on this early activity of the mononuclear phagocyte of the omentum than actual evidence would warrant. Whereas we have noted these phagocytic histocytes within the mesothelium in actual migration, we have never seen them so packed with particles in these situations as they are along the blood vessels. It is probable that by far the major phagocytic activity takes place within the tissue of the organ rather than outside the mesothelial layer. At 3 to 4 hours after injection, the histocytes along the blood vessels are literally packed with graphite; so much so that their nuclear structure is often concealed. In their early phagocytic response these cells may be readily identified by their eccentrically placed nuclei, usually of kidney or horse-shoe shape. At this period, too, fibroblasts, which are usually non-phagocytic, or only slightly so, have engulfed many graphite particles. Figure 2 shows the extent to which absorption by the portion of the omentum within the pouch has progressed. The major routes of blood vessels appear black,

not due to graphite within the blood vessels but to the heavily-laden histocytes and free graphite particles which have accumulated around them. Although the graphite is closely packed around the blood vessels, we have never identified a single graphite-laden cell or free graphite particle in the blood stream of the omentum in the early periods. Figure 3, the omentum of a dog, represents rather accurately the relations which maintain between the circulation of the blood and the graphite-laden histocytes at 3 to 6 hours after peritoneal injection. The peritoneal portion of the omentum in an animal 3 hours after an injection was far less heavily infiltrated than the portion within the pouch, and yet an abundance of black material could be detected grossly, up to and including its attachment to the spleen. Sections show that the graphite hitherto at the surface is now within the mesothelial layers and is largely contained within phagocytes, although much free graphite is profusely scattered throughout both the ascending and the descending limbs of the omentum.

The rate of absorption through the isolated omentum is by no means constant for all animals. A cat killed at 6 hours following an injection may not show greater absorption than one killed at 3 or 4 hours, so that individual variations may occur. This, however, may be due in part also to operative procedures. In general, exploration 3 to 6 hours following a graphite injection into the subcutaneous pouch will show absorption by way of the omentum sufficient to blacken grossly the mesothelial structures related to the spleen and the pancreas inside the abdominal cavity.

Following these early stages the animals were allowed to live for longer periods after the injection had been made into the subcutaneous pouch. Studies were made of the routes of drainage and the site of the absorbed graphite at 12, 20, 24, 36, and 48 hours after injection. Without giving exact details for each animal, we wish to report the sequence of absorption and drainage which these experiments have demonstrated.

In each successive experiment, all portions of the greater omentum and its normal attachments within the peritoneal cavity were pro-

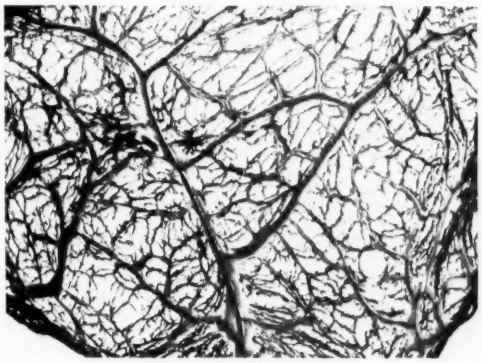

Fig. 3. The omentum of a dog, 6 hours after an injection of graphite into the peritoneal cavity.

gressively more heavily infiltrated (Fig. 4). In the descending limb of the omentum, the color deposits may be readily traced around the pancreas and thence into the hepatoduodenal ligament. Furthermore, the gastrolienal ligament becomes progressively more heavily infiltrated and frequently considerable deposits of graphite are observed along the greater curvature of the stomach, especially toward the duodenum. Within the area of the liver, the gastrohepatic ligament (Fig. 5) and the duodenohepatic ligament are heavily infiltrated with black particles. Likewise that portion of the lesser omentum which covers the caudate lobe of the liver is invariably stippled with pigment. The black particles are not on the outside of these ligaments and investing omentum. Sections show that they are definitely beneath the mesothelial layer and occur either as free particles or in wandering histocytes. We have never positively identified any channels of any sort through which the graphite passes in these hepatic ligaments. Microscopically, there seems to be a diffuse distribution of the graphite granules. Forty-eight hours after an injection it is easy to follow the course of the black granules from the hepatic ligaments and the lesser omentum to the coronary ligament of the liver and thence to the central tendon of the diaphragm. We have not studied sections of the coronary ligaments, and yet they are invariably speckled black, and, as far as our observations go, represent the only demonstrable course whereby

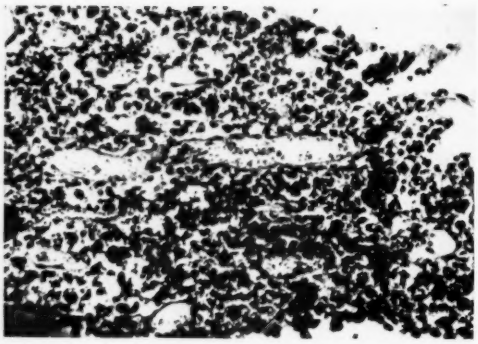

Fig. 4. The peritoneal portion of the great omentum 48 hours after an injection into the pouch. Large numbers of histocytes may be noted. The blood vessels shown are devoid of free graphite or graphite-laden cells (× 400).

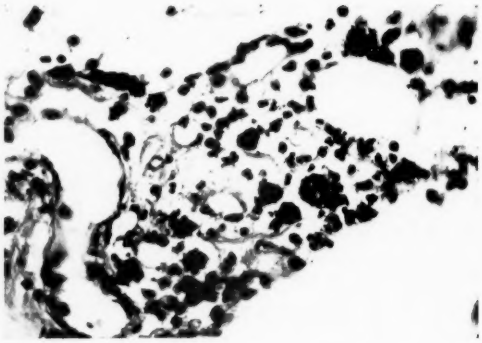

Fig. 5. The gastrolienal ligament, 48 hours after an injection into the subcutaneous pouch. The blood vessels are devoid of graphite (× 500).

the graphite may reach the diaphragm. The peritoneal cavity is always clear, and we are unable to explain the presence of graphite in these structures on any basis except one involving a direct continuity of drainage from the greater omentum to the diaphragm.

The peritoneal surface of the diaphragm in a cat killed 48 hours after a graphite injection into the subcutaneous pouch, presented a picture somewhat similar to that encountered when particles were injected directly into the peritoneal cavity. Strands of black granules, accumulated between adjacent bundles of muscle radiated from the central tendon toward the costal margins of the diaphragm. Sections of the diaphragm showed that these particles were beneath the mesothelial surface massed in the intermuscular spaces, some within the subserous lymphatic plexus and some without. Free graphite was rarely, if ever, seen in a serosal cell of the diaphragm. The evidence leads us to conclude that free particles, as well as phagocytic histocytes, work their way, assisted by circulating fluids, under the mesothelial surface and thence into the extensive lymphatic plexus of the diaphragm.

The passage through the diaphragm is unquestionably by way of the lymphatics, and we have demonstrated graphite in the collecting channels on the pleural surface, in the sternal lymph tracts, and in the anterior mediastinal lymph nodes. From the central tendon of the diaphragm, therefore, the drainage routes toward the anterior mediastinum are identical with those in absorption directly from the intact peritoneal cavity.

COMMENT

These studies support very definitely the hitherto recorded observations that the great omentum readily absorbs and removes foreign particulate material. They further show that its secretory activity, and its adhesive and absorptive functions are by no means severely impaired when the omentum is withdrawn from the peritoneal space and carefully isolated within a pouch in the ventral abdominal wall.

Our observations are not in accord, however, with other recorded observations concerning the manner in which these absorbed materials are removed. The lymphatic distribution of vessels within an organ is the system to which the function of absorption and conduction is usually attributed. The existence of a lymphatic vessel system or lymphatic vessels lined by common endothelium within the great omentum of the adult animal continues to be a question of uncertainty. Ranvier (1896) demonstrated lymphatic channels in the great omentum of newborn kittens, but he maintained that they disappeared before the animal reached adult age. Lymphatic vessels have been described in the omentum of infants. Subsequently, scientific opinion differed and today considerable literature is available, which attempts to confirm or

disprove the existence of a lymph-draining system within the great omentum. The demonstration of such a system is exceedingly difficult, since numerous spaces abound in histological sections which make any adequate interpretation impossible. Suzuki (1910) concluded that the omentum regularly possesses a rich supply of lymphatics. Koch (1911), studying both normal and pathological material, supported Suzuki in his contention. Broman (1914) and Crouse (1915) likewise attributed lymphatics to the omentum. Casparis (1918), using the silver nitrate preparation methods, disclosed extensive lymphatics along the larger blood vessels of the omentum in rabbits, cats, dogs, and man. On the other hand, Shipley and Cunningham (1916) and Seifert (1923) were unable to demonstrate lymphatics in the omentum. Marchand (1924) and Poynter (1928) stated that lymphatics are present only in fetal life and for a short time after birth, when they soon disappear. Seifert (1927), however, stated that while he was not able to demonstrate lymphatics in the free portion of the fetal omentum, he could discern them along the attachment to the greater curvature of the stomach.

Our study is not essentially concerned with the presence or absence of lymphatics of the omentum. We were more concerned with the question of fixation of injected foreign particles and especially with their removal to other regions of the body. Although we have made no attempt to disclose lymphatics in the omentum, our observations lead us to conclude that the removal of the absorbed particles is essentially a function of the lymph drainage rather than the blood vascular system. In each animal experiment, we have studied sections of the great omentum which were made from the portion isolated within the subcutaneous pouch and that within the peritoneal cavity, but we have never been able to demonstrate any graphite particles in the blood stream. Furthermore, we have studied fixed sections of the liver of the cats, shortly after injection of graphite and as late as 48 hours after injection, and we have failed to identify the black granules within the phagocytic cells. The von Kupffer cells are actively phagocytic and, should the graphite have entered the portal vein through the omental circulation, one would expect to identify the particles in these cells lining the sinusoids. We did identify a few mononuclear cells with graphite particles in the sinusoids of the liver of the cat 48 hours after injection, but these were lying free in the blood stream and in our opinion were histocytes that had probably entered the blood stream by way of the anterior mediastinum and the thoracic duct. The fixed or littoral cells were always devoid of the black particles.

Relatively soon following a graphite injection into the pouch, the secretory function of the omentum is early manifested by the abundance of black particles which are firmly adherent to its mesothelium. The extensive vascularity of the organ probably accounts for the large quantities of this serous fluid that is secreted. Within a few minutes these black particles make their way into the omentum where the hitherto inactive histocytes begin their function of phagocytosis. The actual migration of histocytes back and forth through the mesothelium is not frequent, and we are inclined to believe that the more extensive phagocytosis occurs within the omentum and not without. The transfer of the free particles into the omentum is very rapid, and one may only conjecture that a return of a certain amount of the fluid into the omentum carries the particles beneath the mesothelium.

After granules have entered the omentum and phagocytosis has occurred, the histocytes accumulate along the larger blood vessels of the organ. Thus these blood vessels appear black in the omentum removed a few hours after an injection. The lumina of these blood vessels are devoid of either the graphite or the graphite-laden cells, and the endothelium is likewise clear. The histocytes with graphite granules are closely massed along these vessels, and the evidence leads one to conclude that they move along if not in channels, in spaces, surrounding but not connected with the blood vessels (Fig. 6). Occasionally, we have noted in our sections an endothelial pattern or space suggesting a lymphatic vessel both with and without graphite and devoid of erythrocytes (Fig. 7). These areas have been identified in close proximity to blood vessels, and, although

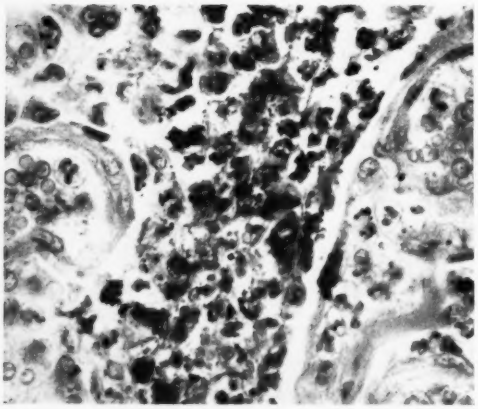

Fig. 6. The omentum of a dog, 6 hours after peritoneal injection. The accumulation of the histocytes and the free graphite around the blood vessels is shown. The blood stream is devoid of graphite particles (× 1000).

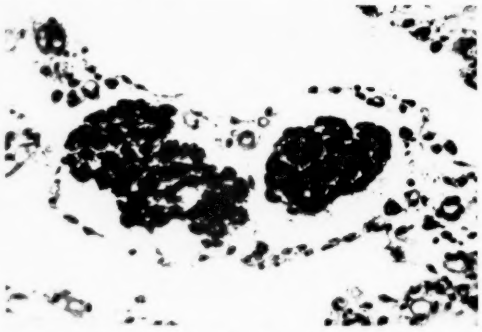

Fig. 7. The accumulation of graphite-laden histocytes into structures that resemble lymph channels in the omentum. Closely related blood vessels are not shown (× 375).

they resemble the lymphatic distribution around the portal vein, we still hesitate to ascribe to them a lymphatic potentiality.

It may be that the conditions within the omentum are not unlike those within lymph sinuses and myeloid tissue. Maximow stated: "As the lymph sinuses are lined with flattened histocytes, so the large venous sinusoids of the myeloid tissue also have a wall, consisting not of common endothelium, but of flattened histocytes, which cannot be separated from the reticular histocytes of the tissue and which show the same functions in an especially high degree, storing of colloidal dyes, phagocytosis of particulate matter, transformation into free macrophages, . . ."

Omitting the details of its early development from the dorsal mesentery, the great omentum is formed of four layers of mesothelium, two comprising the ascending limb, and two the descending limb of the organ. The cavity within this omentum, the lesser sac, largely obliterated in the adult, is continuous with the peritoneal cavity by way of the foramen epiploicum or foramen of Winslow. Thus the peritoneal sac with which we are concerned includes, besides the omental bursa or the cavity of the great omentum, the cavity of the lesser omentum as well. The lesser omentum is a double layer of peritoneum which extends from the lesser curvature of the stomach and the duodenum to the liver, the caudate lobe of which it covers. The duodenohepatic ligament and the gastrohepatic ligament are parts of the lesser omentum. If the two layers comprising the ascending limb of the great omentum are traced forward they enclose the stomach and again unite along the lesser curvature to form the lesser omentum reaching to the liver.

If we are to judge by the distribution of the graphite within the omentum following absorption from the subcutaneous pouch, we must conclude that drainage is to a great extent restricted to the mesothelial layer lining the lesser sac. In tracing the course of drainage around the spleen and to the greater curvature of the stomach by the black deposits in the mesothelium, we have noted that the route to the lesser omentum is always over the dorsal surface of the stomach to the gastrohepatic ligament and not over the ventral surface. Furthermore, the omental covering of the duodenum and the lobe of the pancreas are always heavily speckled with the black pigment, whereas the mesothelial attachment of the colon is less involved. Accordingly, we conclude that the mesothelial lining of the lesser peritoneal sac is the layer more largely involved in the drainage of these foreign particles from the great omentum. The fetal relations of these mesenteries, shown in Figure 8, will clarify our explanation. The caudate lobe of the liver is the only part of this organ ever speckled with graphite, and

this lobe is the only one covered by the lesser omentum. The distinct infiltration of the ligaments of the liver, all a part of the lesser omentum, rather clearly point to a mesothelial conducting mechanism. The circulation of a fluid containing the free particles and the graphite-laden histocytes throughout the extent of the lesser sac are not plausible explanations for the movement of the pigment. The continuity of the lesser sac with the greater peritoneal cavity through the foramen of Winslow would permit a circulation of the granules into the peritoneal space, a phenomenon never encountered.

A further note on the anatomical relations will facilitate an interpretation of the drainage to the central tendon of the diaphragm. If we follow the course of the mesothelium covering the lesser sac, we may observe that it conforms essentially to our pattern of drainage. Starting with the greater curvature, this mesothelial layer covers the dorsal surface of the stomach, and extends from the lesser curvature upward to the liver, covering the caudate lobe. It is now reflected onto the diaphragm and forms the dorsal layer of the coronary ligament. Thence it passes over the dorsal part of the diaphragm to the vertebral column, descends and covers the ventral border of the pancreas and forms the dorsal wall of the lesser sac, and is continued posteriorly to form the inner layer of the great omentum. These anatomical relations rather clearly show how absorption of particulate graphite reaches the diaphragm. As stated, the coronary ligament is always heavily infiltrated with graphite in the animals 48 hours after injection. Anatomically, the ligament is composed of two layers, a ventral layer composed of peritoneum of the greater sac and a dorsal layer, composed of the peritoneum of the lesser sac. The ventral layer of the coronary ligament appears to be less stippled in these drainage experiments than the dorsal layer. This explains, we believe, the absence of graphite in the serosal surface of the ventral portion of the diaphragm which is anatomically a part of the mesothelium lining the greater peritoneal sac. We believe that most of the graphite enters the diaphragm by way of two closely related routes. One of these is along

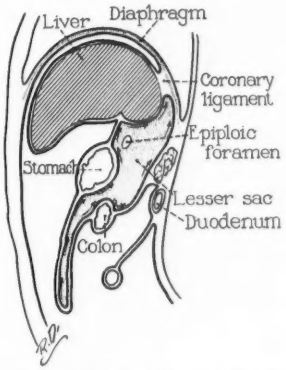

Fig. 8. A diagram of the relations of the fetal omentum to the abdominal cavity.

the posterior wall of the stomach (the anterior wall of the lesser peritoneal sac), thence through the gastrohepatic ligament to the coronary ligament and into the diaphragm. The other route is by way of the pancreas to the crura of the diaphragm, along the posterior wall of the lesser sac, and then into the diaphragm. The radial distribution of graphite, so frequently seen beneath the peritoneal surface of the diaphragm, occurs by migration along the intermuscular fascia from the site of contact of the diaphragm with the superior recess of the lesser peritoneal sac. Thence, the graphite particles enter the subserous lymphatic plexus of the diaphragm, pass through the partition and enter the collecting channels on the pleural surface. Thus the drainage from the great omentum is by way of those lymphatic channels of the diaphragm and the ventral mediastinum, rather than through the cisterna chyli, and the thoracic duct. There are, then, two systems of drainage from the abdomen, one associated with the gastro-intestinal tract passing through the mesenteries to the cisterna, and the other associated with the omentum and the diaphragm, passing through the ventral mediastinum to the cervical lymph ducts.

We do not wish to state that all particles absorbed and fixed by the great omentum are transferred to other parts of the body. We are well aware that the omentum, following absorption of particulate graphite from the peritoneal cavity, will remain black for many

months. We do affirm, however, that a considerable portion of the materials so absorbed, especially the finer particles, may be removed promptly to the diaphragm, and thence to other portions of the body, through a mesothelial conducting mechanism operating by way of the omentum.

SUMMARY

A method is described for the study of absorption by the omentum, isolated from all structures within the peritoneum. Studies have been made on the degree of absorption by the isolated omentum from the subcutaneous pouch at frequent intervals, ranging from 30 minutes to 48 hours after an injection. Lymphatic vessels within the omentum have not been demonstrated conclusively and yet absorption from this organ is essentially by way of the lymphatics of the diaphragm and the mediastinum. It was not possible to demonstrate either free particles or graphite-laden histocytes in the omental blood vessels, following an injection into the subcutaneous pouch. Following phagocytosis of the graphite particles by active mobile histocytes these cells accumulate along the blood vessels and pass toward the gastric and splenic attachments of the great omentum. The routes of drainage from the distal part of the omentum follow essentially the mesothelial lining of the lesser peritoneal sac. From the gastrolienal ligament, drainage follows around the dorsal surface of the stomach, along the lesser omentum to the caudate lobe of the liver, and thence along the coronary ligament of the liver and the central tendon of the diaphragm to the anterior mediastinal lymph nodes.

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POSTCONCEPTION PELVIC IRRADIATION OF THE ALBINO RAT (MUS NORVEGICUS): ITS EFFECT UPON THE OFFSPRING

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THE findings in two recently completed clinical studies indicate that pelvic irradiation employed during pregnancy is very likely to arrest the development of the fetus. The fact that of 76 full-term children irradiated *in utero*, 18 were microcephalic—while still other developmental defects were exhibited by some of the remaining ones—definitely points in this direction.

Experiments upon a variety of the lower animals by different observers (1) confirm these clinical findings, although few of these studies were made upon mammals. Irradiation of fertilized animal ova and of immature animal young appeared to be followed in a large proportion of cases by a rather wide variety of physiological and anatomical disturbances, e.g., early death, weakness, underdevelopment, and very high frequency of gross structural deformities. These disturbances seemed to vary with the kind of animal treated, the amount of irradiation employed, and the time of the treatment in relation to the date of fertilization, and to other factors.

One of the objects of the present study was to check the results of the other observers in this field. The rat was chosen for our experiments because most of the earlier investigations had been concerned with animals lower in the developmental scale, making them less important as a means of evaluating the results of human irradiation.

The desired end of the experiment was to find out whether embryonic irradiation, in non-lethal doses, would or would not be followed by the birth of young exhibiting gross structural abnormalities.

The irradiation was given with equipment of the mechanically rectifying type, energizing a broad-focus Coolidge tube. The roentgen machine was calibrated with a Wulf ionometer, the latter in turn calibrated in Germany with the proposed international "R" unit. The operating factors were as follows: 127 kilovolts (peak): 5 milliamperes: 30 centimeter skin target distance, and 6 millimeters of aluminum. One hundred and eighty milliampere minutes (m.a.m.) of exposure gave 800 R units of intensity.

The animals were treated as shown in Figure 1, strapped to the small concentrically arranged tables. The cephalic half of each animal was protected by a lead plate 2 millimeters in thickness. The Coolidge tube target was directed at the central point of the circular base upon which the small tables were fastened with adhesive plaster, the skin target distance (30 centimeters) being measured from the target to the level of the rats' backs.

With this technique, 120 animals were subjected to from 1 to 6 exposures, varying in strength from 45 to 360 milliampere minutes (200 to 1600 R).

Gestation in the rat consumes from 22 to 23 days. Of the 120 treated animals, 34 cast litters within less than 22 days of their last roentgen exposure, i.e., the embryos in these cases had received at least one roentgen exposure.

The litters of these 34 animals varied in size from 1 to 11, the most common being 2, with an average size of 3.6 young. In a

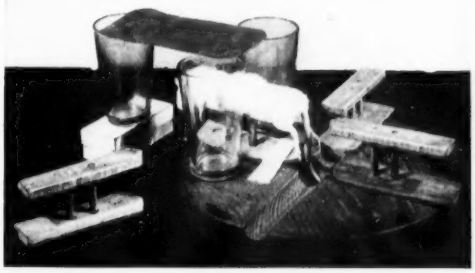

Fig. 1. Showing circular base and 4 concentrically arranged tables, used for the purpose of holding unetherized adult rats in position for uterine irradiation. Circular bands of zinc oxide adhesive plaster, around the upper and lower halves of the body, maintain the animal in desired position and check undue movement. Each table is, in turn, strapped to the circular base. Note the sheet of lead protecting the upper half of the body.

Fig. 2. Showing a rat less than one day old, which had been irradiated while *in utero*. The centimeter rule indicates the degree of magnification employed in taking the photograph. Note the apparent difference between the degree of development of the hind legs and that of the fore legs in respect to the size and shape of the feet and the depth and length of the digital grooves.

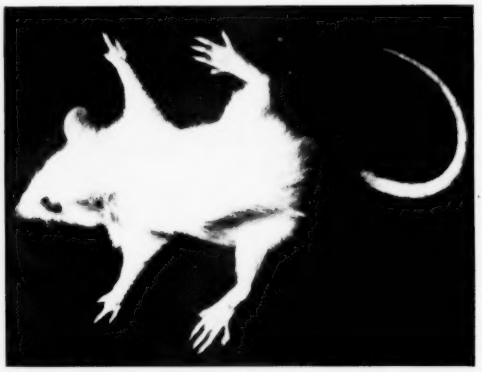

Fig. 3. Showing a rat which had been irradiated while *in utero*. Note the rudimentary development of the second toe on each fore foot. The dark area around the eye is due to a conjunctival discharge.

group of 35 control litters the most common size was 7 young.

Grossly defective young were cast by 5 of the 34 irradiated animals. The number of these young appeared to be in direct proportion to the degree of the irradiation. For instance, of the 90 young born of 20 animals which had received only 45 milliampere minutes (200 R) of exposure, not one was found defective. On the other hand, 12 animals exposed to 90 milliampere minutes (400 R) (twice the above amount) cast 3 litters (one each) containing defective offspring, while each of 2 litters exposed to 180 milliampere minutes (800 R) or 4 times the smallest exposure exhibited one or more defective young. If these 2 latter groups are considered as a single group, it will be observed that of the 14 animals receiving 90 milliamperes (400 R) or more, 5 (a percentage of 35.7) gave birth to defective young.

These litters comprised a total of 13 offspring, of which 2 were known to be alive at birth but died within a few hours. The remaining 11 were either stillborn, or were killed by their parents, or died natural deaths before being observed. Of the whole group of 13 young, 6 exhibited developmental defects of the extremities.

Of the 5 animals casting litters containing defective young, 4 gave birth to a total of 10 offspring, all of which were, at least externally, well formed, except for a foot deformity exhibited by 5 of them.

The foot abnormality exhibited by these 5 young (Fig. 2) was characterized by lack of the normal depth of the digital grooves. In 4 of the 5 animals only the hind feet were affected, while the fifth one exhibited the defect in all 4 feet. In addition, where the digital grooves were absent or distorted, the entire foot was deformed so as to appear clubbed. In a number of instances the end of the extremity was perfectly smooth, similar in appearance to an amputation stump of long standing. In those instances in which only the hind feet were affected, the fore feet appeared to be perfectly normal in every respect.

The fifth litter consisted of only one young, shown in Figure 3. This animal appeared to be perfectly healthy and lived until the end of the experiment. Its fore feet, however, had only 3 well developed toes each, with the normal site of the missing toe marked by a rudimentary bud which suggested an arrest of development.

For a number of reasons it is believed that the abnormalities which have just been decribed, and which are depicted in the two accompanying photographs, are the result of the embryonic irradiation. The clinical findings suggest this as does also the earlier experimental evidence advanced by other workers. In the case of the present experiment, the defects varied (at least in frequency) with the degree of the embryonic exposure. The same defect was observed several times in the same

litter and in more than one litter. Furthermore, no such similar defects were observed in more than 600 control young examined during the previous 2 years. In addition, Dr. Helen Dean King of the Wistar Institute of Anatomy and Biology (from which Institute the mother animals were secured) reports that she has never observed any such defects in the more than 125,000 rats recorded in her laboratory.

SUMMARY AND CONCLUSIONS

1. The litters of 34 female albino rats, irradiated when pregnant, have been studied.

2. In 5 of these 34 litters one or more young exhibited either clubbing of feet or absence of toes.

3. The frequency with which defective young were produced appeared to vary directly with the degree of exposure.

4. Though no definite conclusion can be drawn from the observations set down in the present paper, due largely to the scantiness of material available for analysis, it is significant that the deformities observed among the young of animals irradiated when pregnant have not been duplicated in a series of 125,000 non-irradiated control animals.

The authors are very greatly indebted to Mr. J. L. Weatherwax, physicist of the Philadelphia General Hospital, for his kindness in calibrating the roentgen machine.

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THE CIRCULATORY COMPLICATIONS OF PROSTATECTOMY

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ANY operative procedure upon a patient during the sixth decade of life or thereafter is undertaken in the face of a diminished circulatory reserve. In the younger individual, in the absence of cardiovascular disease, the circulation, with the aid of its compensatory mechanism, is adequate to meet the most varied demands. With advancing age, however, coincident with degenerative changes in the blood vessels and myocardium, its functional reserve gradually decreases. The arteries become more or less sclerosed; irregular plaques are formed in the intima and may involve the medial coat. Diminution in the caliber of the vessels and impairment of the elasticity of their walls result. The changes in the myocardium are somewhat more varied. In the individual fibers histological alterations, pigmentation, occur which make it possible to distinguish an old fiber from a young one. Furthermore, the myocardium may show hypertrophy or fibrosis. The first is the normal response to the increased work required to maintain an adequate blood supply through narrowed, inelastic arteries. Unless the valves are damaged or, due to emphysema, the resistance in the pulmonary circulation is increased, this enlargement is confined to the left ventricle. Secondarily, the aorta dilates, chiefly in the region of its base and arch. The second change, myofibrosis, is due to one or both of two factors: first, to the normal increase in interstitial connective tissue and wasting of muscle fibers, which occur with age; second, to localized or diffuse muscular degeneration and its replacement by fibrous tissue incident to impairment of the blood supply to the myocardium. The latter is augmented by any sclerotic narrowing of the coronary arteries either at their origin in the aorta or along their course.

Whatever may be the anatomical results of such changes the inevitable physiological effect is an impairment of the capacity of the heart to meet increased demands. The heart's efficiency depends upon the integrity of its metabolism; this presupposes an adequate blood supply to the myocardium in proportion to the load placed upon it. The circulatory requirements under normal conditions can, in the majority of cases, be supplied, but unusual demands (overexertion, fever, hypertension, etc.) cannot be borne without some evidence of circulatory insufficiency.

Physical examination of the senile heart frequently presents the following clinical picture: the impulse is often obscured by overlying emphysematous lung, and when visible or palpable it is usually displaced somewhat to the left. The relative cardiac dullness is enlarged, particularly to the left and downward, and the retrosternal dullness, in the first and second interspaces, is widened, corresponding to the dilated aorta. The heart sounds at the apex are distant and feeble, and are often accompanied by a blowing murmur (functional mitral insufficiency). Over the base the aortic second sound may be sharp but, in the absence of hypertension, is not greatly accentuated. More characteristic is a systolic murmur in the second right interspace, sometimes transmitted upward. This is produced, supposedly, either by arteriosclerotic stiffening of the aortic cusps or by the relative disproportion in the diameter of the normal aortic orifice and the dilated arch. The cardiac rhythm may be normal but is often interrupted by ventricular extra systoles. Rarely auricular fibrillation may be present.

In the absence of myocardial insufficiency, such patients present few symptoms referable to the heart. They are conscious of limitation of their capacity for physical work, and occasionally complain of palpitation; this latter symptom is sometimes lacking even in cases showing numerous extrasystoles. As the circulation becomes overtaxed, however, more obvious symptoms develop: fatigue, breathlessness while talking, dyspnœa on slight exertion, and orthopnœa. Finally a small proportion of these patients complain of typical

anginal pain. The physical signs of myocardial insufficiency may be present in varying degrees. These include evidence of pulmonary œdema, (râles at the lung bases), hepatic enlargement, and œdema of the extremities.

During the past 3 years at the request of Dr. Young we have studied the various circulatory problems encountered in patients undergoing prostatectomy in the James Buchanan Brady Urological Institute of the Johns Hopkins Hospital. This operation is most often necessary at an age at which the changes described above are well advanced. Urinary obstruction, which it is calculated to relieve, carries with it a series of strains upon the circulation, and some of the postoperative complications are prone to bring about myocardial insufficiency. Such obstruction, with the resultant impairment of renal function, produces a condition which, at the time, may be indistinguishable from chronic nephritis with nitrogen retention and hypertension. With the relief of the obstruction these disappear—unless, as in some cases, chronic nephritis and urinary obstruction coexist—but until then the systolic discharge of the heart is opposed by an increased resistance, alone sometimes sufficient to cause symptoms of myocardial failure.

A factor of considerable importance in contributing to circulatory failure is the interference with adequate rest. Frequency of urination, particularly as it occurs at night, may so disturb sleep that the patient becomes wellnigh exhausted. Infection, even though localized to the genito-urinary tract, throws an additional burden upon the circulation, particularly if it be accompanied by fever.

Pre-operative care. It has become increasingly apparent that the frequency and severity of postoperative cardiac complications may be lessened by adequate pre-operative preparation. *Rest* is a most important pre-operative measure. To this end, as well as to relieve the frequency of voiding and to restore the renal function, catheter drainage should be established. Sedatives, luminal, or even opiates, should be employed when necessary. In many cases cystoscopy must be delayed until adequate rest has been obtained.

Fluids. Urinary obstruction with its resultant functional renal impairment and nitrogen retention necessitates the administration of large quantities of fluid. And yet any degree of congestive heart failure is to be regarded as an indication for restricting the fluid intake. When both conditions are encountered one can steer only a middle course. It is impossible to follow the same scheme for every patient. The degree of circulatory insufficiency must be determined by careful examination in each case, and the fluid limit established accordingly. In our experience it is unwise to force fluids at once in large amounts, following the patient's admission, if signs of myocardial insufficiency be present, but more advisable to increase gradually the fluid intake after 1 or 2 days' rest and digitalization. In general, too, in any patient showing signs of myocardial failure, the administration of fluids by the intravenous route is to be undertaken with caution. Recently we have adopted the following method:[1] A large transfusion needle is inserted into one of the veins in the forearm and strapped in place by adhesive so that it will not slip out. The arm is then so fastened to a board that the patient cannot bend the elbow. Through the needle normal salt or 5 per cent glucose is injected continuously by a drop method quite similar to the Murphy drip and so regulated that the patient receives not more than 100 to 200 cubic centimeters an hour. In this way large amounts of fluid may be given so slowly that the heart is not embarrassed by the increase in volume of the circulation.

What are the danger signals? How can one know that the limit of fluid tolerance is being reached? This again must be decided for each case but the most valuable sign is an increase in the number and extent of râles at the lung bases. In many such cases moist râles are audible at the lung bases on admission but are usually scattered. When large numbers are audible over the lower back on both sides fluids should be restricted to 2,500 cubic centimeters or less, depending upon the urinary output.

Diet. Patients with œdema should receive a diet poor in salt—1.0 gram or less daily. In cases with impaired renal function the

[1] A similar method was described by Matas. Ann. Surg., 1924, lxxix, 643.

tention of salt may contribute to the accumulation of œdema.

Hyperventilation. With advancing age the thoracic cage becomes more and more rigid as the costal cartilages ossify. At the same time the lungs themselves undergo characteristic changes. The alveolar septa waste and the alveoli coalesce, producing the so-called senile emphysema. The residual air is increased, the vital capacity is diminished and expansion is limited. Upon auscultation the breath sounds are distant. If the patient has been recumbent prior to examination, dry, crackling râles are audible at the end of inspiration. These are due to the reopening of alveoli compressed in the recumbent position and are to be distinguished from moist râles which are pathognomonic of pulmonary congestion. The circulation in the lung is impeded by any considerable degree of atelectasis and is enhanced by normal pulmonary ventilation. Aside from its effect in reducing the incidence of postoperative pulmonary infection, as reported by Scott[1], hyperventilation has been found effective in relieving hypostatic congestion, both pre-operative and postoperative, in older individuals. This may usually be accomplished voluntarily by the patient; in some cases it must be induced after operation by causing him to inhale a mixture of 5 per cent carbon dioxide in oxygen.

Digitalis. As has been so often stated, the indication for digitalis is myocardial insufficiency; the routine administration of this drug is not only useless but unwise. It should, however, be given to any patient showing signs of congestive failure, and in adequate quantity, i.e., 1.5 grams standard leaves per 100 pounds body weight, or an equivalent amount of some standardized preparation. This is best administered in divided doses over a period of 48 hours or more, except in cases of acute failure, to which it may be given more rapidly. After the maximum therapeutic effect has been obtained, digitalis should be continued at 0.1 to 0.2 gram daily to replace the amount normally excreted. If the patient has received this drug prior to admission digitalis must be administered in smaller doses and a longer period allowed for digitalization in order to avoid intoxication. The development of acute dilatation calls for an increase in digitalis dosage, or, if the patient has not been fully digitalized previously, for strophanthin (0.5 to 1.0 milligram intramuscularly). In cases showing numerous ventricular extrasystoles the combination of digitalis with dionine may often restore the normal rhythm more quickly than does digitalis alone. This is given in capsules (folia digitalis 0.1 gram, dionine 0.006 gram) and continued up to the therapeutic maximum for digitalis.

Operation. The circulatory strain resulting directly from an operation is due to one or both of two factors. The first is reflected in a reduction of the vital capacity, and is particularly marked in abdominal operations. The normal vital capacity falls steadily during the sixth and seventh decades of life and after, until at 80 it is but 45 to 50 per cent that of the normal at 30 years of age. Perineal operations have been shown to be accompanied by little or no reduction in the vital capacity,[2] a fact which we have repeatedly confirmed. As regards the perineal operation, the chief circulatory strain may be due to the second of these factors, i.e., the anæsthetic. Here the choice is often a nice one. Aside from increasing the frequency of postoperative pulmonary complications, general anæsthesia, ether or nitrous oxide, is attended by a degree of anoxæmia and secondary circulatory changes which must be regarded as dangerous in elderly patients. Herein lies the advantage of caudal or spinal anæsthesia. Except in occasional, very apprehensive, patients with symptoms of angina pectoris, we have used caudal or epidural anæsthesia, injecting 20 cubic centimeters of a 3 per cent solution of procaine into the sacral hiatus. Morphia (16 milligrams) is given the night before operation to insure rest and again just before operation. Caudal anæsthesia is attended by a fall in blood pressure varying from 10 to 50 millimeters mercury in about 50 per cent of the cases, by a rise of 10 to 30 millimeters in 30 per cent, and by no pressure change in the remainder.

Postoperative treatment. Digitalis should be continued after operation in doses just sufficient to maintain the therapeutic effect

[1] Scott, W. J. M. J. Am. M. Ass., 1929, xciii, 101.

[2] Powers, J. H. Arch. Surg., 1928, xvii 304.

(0.1 to at most 0.2 gram daily). During the first 2 days after operation this is usually best administered by injection—digifoline 1 to 2 cubic centimeters. Hyperventilation is a most important postoperative measure. The interference with respiration caused by abdominal distention may sometimes contribute to circulatory failure. It should, therefore, be prevented as far as is possible and vigorously combatted as soon as it appears. Certain drugs, and particularly morphine, contribute to distention by relaxing the intestine. Schlesinger's solution and pantopon are less liable to foster distention and at the same time are efficient sedatives. After prostatectomy any treatment per rectum—enema or passage of rectal tube—is to be avoided on account of the danger of embolism. Turpentine stoupes, pituitrin or eserine are usually effective in dispelling distention. To avoid hypostatic pulmonary congestion these patients are turned frequently in bed.

Uncomplicated cases are permitted to sit up in a chair for a half hour on the fourth day after operation. Activity is gradually increased and supplemented by, at first light, and then more vigorous massage. When first allowed up many of these patients develop œdema of the feet, ankles, and lower legs. This is particularly true of those who have, for one reason or another, been confined to bed for some time. In the majority of instances this is an evidence of local, rather than general, circulatory insufficiency. The return of venous blood from the lower extremities is, in no small measure, dependent upon the tone of the leg muscles. As this develops, the œdema often disappears. Massage and graduated exercises contribute to this. In an occasional case it is necessary to provide elastic stockings to be worn while walking during the early days of convalescence.

The most alarming postoperative complication is acute cardiac dilatation. In this condition the myocardium becomes suddenly unable to accomplish an adequate systolic discharge. Symptoms and signs of failure rapidly develop, the patient becomes cyanotic, the pulse rapid and thready, the blood pressure falls, the pulse pressure is reduced, pulmonary râles become evident and the liver is engorged. The heart is often demonstrably enlarged, the sounds feeble, and a gallop rhythm is frequently audible. The cause of dilatation is not always evident immediately. It is theoretically due to increase in the myocardial load beyond the optimum limit in proportion to its blood oxygen supply.

The most common factors causing acute cardiac dilatation are pulmonary embolism and coronary occlusion. The incidence and results of the former have recently been reported by Thomas and Alyea.[1] The abrupt blocking of a greater or lesser portion of the pulmonary circulation suddenly increases the resistance to the output of the right ventricle. The added anoxæmia contributes to the failure of the myocardium by impairing its oxygen supply. Coronary occlusion, by depriving a portion of the myocardium of its arterial supply, may bring about acute and alarming symptoms of dilatation. If the occlusion involves a large vessel, death is the inevitable result. If, however, it occupies one of the terminal branches, compensation may later be restored. Coronary occlusion is attended by symptoms and signs of cardiac dilatation and by moderate fever and leucocytosis.

The patient is often conscious, restless, and extremely apprehensive. A sedative, usually morphia, should, therefore, be given at once. If the patient has not been previously digitalized this should be accomplished as rapidly as possible, best of all with strophanthin. The fall in blood pressure is due only in part to peripheral vascular relaxation; it is the result chiefly of the decreased systolic discharge on the part of the heart. For this reason adrenalin should be used cautiously; it may only increase the load upon an already overburdened ventricle. Caffeine is, under these conditions, a far more rational stimulant than adrenalin.

Finally an abnormal rhythm, auricular fibrillation, or, more rarely, flutter, may so interfere with the output of the heart as to cause symptoms of circulatory failure. This may be present upon admission or may develop suddenly following operation. The ventricular rate can be controlled with

[1]Thomas and Alyea. South. M. J., 1929, xxii, 737.

adequate doses of digitalis. In the case of flutter the rhythm may be restored to normal by means of quinidine after digitalization.

CASE REPORTS

G. P., aged 77 years, admitted to the hospital December 4, 1928, complaining of urinary frequency and urgency which had been present for 4 years. Three years ago he began to have pain over the upper precordium and in the left upper arm following exertion or a heavy meal. Examination in October, 1926, revealed benign prostatic hypertrophy with residual urine 400 cubic centimeters. He often felt faint but he never lost consciousness. The diagnosis at this time was arteriosclerosis, cardiac hypertrophy and dilatation, and angina pectoris. Blood pressure was 170–90. These symptoms persisted up to time he was last admitted to the hospital.

Physical examination disclosed a large man with arcus senilis, lungs clear throughout. Heart rhythm was regular, rate 80. Impulse was visible in fifth left interspace 11.5 centimeters from midline, and was localized and forceful. Relative cardiac dullness measured in first interspace, 3.0 centimeters left, and 3.5 centimeters right; in second interspace, 3.0 centimeters left, and 3.0 centimeters right; in third interspace, 6.5 centimeters left, and 3.0 centimeters right; in fourth interspace, 11.0 centimeters left and 4.0 centimeters right; in fifth interspace, 13.0 centimeters left, and 4.5 centimeters right. At apex, sounds were loud, and there was a faint systolic murmur. Over mid and upper portions of the precordium there was another systolic murmur, loudest over the aortic area, and transmitted upward into neck vessels. The second aortic sound was loud and ringing. The retinal arteries were narrowed and tortuous. The radials and brachials were palpably thickened. The liver was not enlarged. No œdema was present in extremities. *Impression.* Benign prostatic hypertrophy, arteriosclerosis of peripheral and coronary vessels, dilatation of aorta, cardiac enlargement, angina pectoris.

Patient was prepared for operation over a two-week period with rest and digitalis. He got out of bed once and fainted. Blood pressure taken immediately thereafter was 130–80. Perineal prostatectomy was performed December 18, 1928, by Dr. Young. Ether anæsthesia was used. Systolic blood pressure during operation varied from 150 to 180. Immediate convalescence was uneventful.

Three weeks after operation patient awakened at night with pain in his left arm. He insisted upon arising next day. That evening he complained of constant, dull pain in the upper precordium and left arm; he became apprehensive. Examination showed slight cyanosis and the heart not demonstrably larger than before. Sounds were faint, the apical systolic murmur louder; a presystolic gallop rhythm was audible over the entire precordium. Liver edge 2 centimeters below the costal margin. Patient was confined to bed, sedatives were administered, and digitalis dosage increased for 5 days. Gallop rhythm and pain gradually disappeared. During the week following this attack patient had a fever of 100 to 101 degrees and leucocytosis, 10,000. He remained in the hospital 3 weeks after the cardiac attack and then rested at a hotel in the south. Massage and graduated exercise were begun after 2 weeks and were continued after his discharge from the hospital. He returned 4 months later. At this time the physical examination showed the same conditions as were present when he entered the hospital for the first time.

This case illustrates the occurrence of minor coronary occlusion in a patient who had had symptoms of angina pectoris for several years. Operation was undertaken with considerable apprehension and then only after 2 weeks' preoperative treatment and because it seemed absolutely necessary. The choice of ether anæsthesia was made for two reasons: (1) The history of syncopal attacks and the observation of one such associated with a fall in blood pressure, which made it seem unwise to risk the lowering of pressure frequently associated with caudal or spinal anæsthesia; and (2) the excitable temperament of the patient. It was felt that, during an operation under local anæsthesia, this latter might cause a dangerous rise in pressure.

D. G., aged 76 years, admitted to the hospital in March, 1928. His complaint was urinary frequency of 10 years' duration. Urinary retention was noticed 6 years prior to admission; during that period the patient catheterized himself. A large vesical calculus was discovered by cystoscopy in January, 1928. Patient was short of breath and had precordial discomfort upon exertion; cramps in the lower legs were brought on by walking. He gave no history of angina pectoris.

Physical examination disclosed a pronounced arcus senilis and clouding of the lens in each eye. Respiratory movements were limited by the rigid thoracic cage, respiratory rate 28 per minute. The percussion note was resonant over both lungs and over the upper portions it was tympanitic in quality. Fine moist râles were audible over both bases. Examination of the heart showed impulse visible 9.5 centimeters from the midline in the fifth left interspace. Relative cardiac dullness: first interspace, 2.5 centimeters left, 2.5 centimeters right; second interspace, 3.0 centimeters left, 2.5 centimeters right; third interspace, 4.5 centimeters left, 3.0 centimeters right; fourth interspace, 8.0 centimeters left, 4.0 centimeters right; fifth interspace, 11 centimers left, 4.0 centimeters right. Heart sounds were distant. Systolic murmur at apex was transmitted outward; over the aortic area the systolic murmur was trans-

mitted upward. Ventricular extrasystoles occurred every third cycle. Blood pressure on admission was 200–100. Blood urea 0.64 grams per 1. Vital capacity on admission was 800 cubic centimeters. All superficial arteries were palpably thickened, and radial arteries contained calcified plaques in their walls. The retinal arteries were irregularly narrowed. Liver edge palpable 1 centimeter below the costal margin. Slight œdema was noticeable over both ankles and tibiæ.

The bladder was drained with a retention catheter; 4,000 cubic centimeters of fluid were administered daily; patient was kept in bed except for 2 hours daily in a chair. He received 12 doses of digitalis leaves 0.1 gram and dionine 0.006 gram within the first 3 days. Digitalis leaves were then continued, 0.2 gram daily. Upon the twelfth day after admission the blood pressure had fallen to 160–75 and blood urea to 0.40 gram per liter. Vital capacity at that time was 1,000 cubic centimeters. The liver had decreased in size; œdema of ankles had disappeared. The respiratory rate was 16 per minute. The excretion of phthalein rose from 60 per cent to 80 per cent over a 2 hour period. The ventricular extrasystoles were still present.

Perineal prostatectomy was done by Dr. Young, caudal anæsthesia being used. Digitalis was continued at 0.1 gram daily. The patient was confined to bed for 4 days and then allowed to sit up for increasing periods during the subsequent 10 days.

This case presents the problem involved in a patient with advanced arteriosclerosis, moderate hypertension and a low vital capacity who required removal of the prostate for the relief of urinary obstruction. Upon admission there was evidence of myocardial insufficiency. This disappeared as did also the hypertension, under pre-operative treatment of 12 days' duration. The patient underwent operation without any circulatory complication.

E. S., aged 72 years, was admitted to the hospital June 16, 1928. He had suffered from frequency and difficulty of urination for 6 years. He had been frequently catheterized during the last 5 years. Breathlessness was noticed on exertion; but there were no symptoms of angina pectoris. The extremities were not œdematous. The lungs were clear on percussion and auscultation. The cardiac borders were within normal limits; there was no cardiac enlargement; the sounds were of good quality, no murmurs, and rhythm was normal. Blood pressure was 140–90. Urological examination showed a benign prostatic hypertrophy. On June 18, 1928, perineal prostatectomy was done by Dr. Young, caudal anæsthesia being used. An attack of acute epigastric discomfort on the ninth day after operation was followed by pain in lower right axilla, accentuated by deep inspiration. Pain was relieved by sedatives and strapping of chest. The next morning movements of right side of the chest were limited on inspiration; there was a small area of dullness in right lower axilla with suppression of breath sounds over this region. No râles and no friction were noticed. Diagnosis: Pulmonary infarction. X-ray examination showed infiltration and pleurisy at the right costophrenic angle. Three days later pain was again noticed in the right chest. Temperature rose to 102.2 degrees and for the next 5 days ranged from 99 to 101 degrees. After the second attack of pain, examination showed a large increase in the area of dullness in the right lower lobe. Patient was disoriented and weak, blood pressure was 100–75, with gallop rhythm at apex. Three days later bright red blood appeared in the sputum. Cardiac rhythm was now irregular. Patient received 1.2 grams of powdered digitalis in 2 days and 0.2 gram each day following for 2 weeks. X-ray examination 2 days later showed pneumonia at the right base. Blood streaked sputum persisted for 2 weeks. Temperature gradually fell to 99 degrees. White blood count was 11,400. Signs of thickened pleura at the right base were noticeable for a month. After the patient had sat up in chair his ankles became markedly œdamatous. Cardiac rhythm continued to be entirely irregular, pulse deficit 50 per cent. There was no œdema of the lungs and œdema of the extremities disappeared in 1 week. An electro-cardiogram showed an auricular flutter. Patient was given 1.0 gram digitalis in 24 hours, followed by 0.8 gram quinidine. The next day a normal sinus rhythm supervened. A week following this the patient was discharged from the hospital with a normal sinus rhythm, no œdema of the extremities or of the lungs.

In this case convalescence was interrupted by pulmonary infarction, acute cardiac dilatation, and auricular flutter. With absolute rest, sedatives, and digitalis, the patient recovered from the effects of the pulmonary infarction and the signs of circulatory collapse disappeared. The rhythm of the heart reverted to normal under quinidine.

SUMMARY

1. Hypertrophy of the prostate, requiring operative removal, usually develops at an age at which the functional circulatory reserve is already diminished.

2. Proper pre-operative care, the use of caudal anæsthesia, and careful postoperative observation do much toward avoiding circulatory complications of prostatectomy.

The authors are indebted to Dr. Hugh H. Young for permission to study these cases on his service.

SIMPLE, NON-SPECIFIC ULCER OF THE COLON

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SIMPLE, non-specific ulcer of the colon is an ulcerating lesion which is not due to the action of any specific organism such as specific ulcer of tuberculosis, syphilis, ulcerative colitis, dysentery, typhoid fever, or to the local action of any chemical agent and which is not secondary to or above a malignant tumor causing constriction.

In September, 1928, I published a paper entitled "Simple, Non-specific Ulcer of the Colon." The article was a study based on 50 cases collected from the literature and observations on 3 personal cases.

I directed attention to the fact that simple ulcer, analagous to gastric or duodenal ulcer, may occur on any part of the alimentary canal from the œsophagus to the rectum and that the gross and histological characteristics of this lesion are similar in every way to the so-called peptic ulcer.

Ulcer of the stomach in association with ulcer of the duodenum, or in association with ulcer of the jejunum, ileum, or colon, in the same patient has been observed by some investigators (2, 3, 4). In the series which I studied the combined lesions, ulcer of the stomach and of the colon, occurred in 4 cases.

Since the presence of ulcers occurring both in the stomach and duodenum in the same patient is not rare and we are ready to accept the etiology in both ulcers to be the same, it is rational to believe that simple ulcers occurring in the stomach and in other segments of the gastro-intestinal tract in the same patient have a common etiology. Because of my findings and the observations of others where the lesions occurred both on the stomach and colon, I am led to believe that there is probably a common etiology. If this is true, it would seem that simple ulcer of the colon must be a manifestation of the same general disease as peptic ulcer, and therefore we must look to causes other than local for the production of these lesions.

Of 53 cases that have been reported in literature since 1837, the pre-operative diagnosis of appendicitis was made 11 times, first appearing in literature in 1910. Since 1910 there have been 23 cases reported, of which number the diagnosis of appendicitis was made 11 times—almost 50 per cent. In 10 of the 11 cases, the ulcer occurred either in the cæcum or the ascending colon. The diagnosis of ulcer, when the lesion occurs in the right half of the large bowel, is very difficult to differentiate from that of appendicitis. Recently I saw and operated on another patient with a perforating ulcer of the cæcum where a diagnosis of appendicitis was made.

Mrs. C. P., female, age 61 years, white, entered the Surgical Service of the Beth Israel Hospital on April 5, 1929, with a complaint of severe pain in the right lower quadrant. Her family and past history were essentially negative except for mild diabetes mellitus for past 6 months. About 1 week ago she began to have pain in right lower quadrant. Pain gradually became worse and continued for about 1 week being moderately severe until day of admission when suddenly the pain became much more severe. There was no nausea or vomiting. There was no elevation of the temperature or increase of pulse rate until today when there was a slight elevation of temperature. The bowels were regular with catharsis and she had a bowel movement today. She has had a chronic cough for many years but with no expectoration. Menopause occurred 12 years ago—there is no dysuria or hæmaturia. Patient has been on insulin and regulated diet. There never has been any jaundice and there was no history of any previous digestive trouble.

Physical examination disclosed well developed, obese woman of 61 years, lying in bed complaining of considerable pain in the right lower quadrant. The skin was moist and warm and the face flushed. There was nothing else of note except marked tenderness over the right lower quadrant and some spasm and rigidity of the muscles in this region. The costovertebral angles were not tender—abdomen was tympanitic throughout—no definite mass could be made out over the tender area. A provisional diagnosis of acute appendicitis was made and immediate operation was advised. The results of urine analysis were negative. White blood cells 11,000. Temperature 100 degrees F., pulse rate 100, respirations 24. Blood pressure: systolic 155, diastolic 90.

Operation was done April 5, 1929. Under local anæsthesia a McBurney incision was made. There escaped a slight amount of a seropurulent exudate.

The cæcum was found indurated and adherent to the parietal peritoneum. The vermiform appendix was found to be much smaller than normal, almost obliterated, and could not possibly have accounted for the inflammatory reaction within the peritoneal cavity and the patient's clinical condition. It was removed, however, in the usual manner. Since the cæcum was found to be indurated and adherent it was impossible to continue the operation without the aid of a general anæsthetic. On account of the patient's condition nitrous oxide and oxygen was used. The cæcum was then freed and an indurated mass about the size of a silver half dollar found in the posterior lateral aspect. The induration corresponded to an isolated ulcer, the center of which revealed a perforation about the size of a lead pencil in diameter from which was escaping intestinal contents. There was no evidence of any other ulcer or enlarged glands and it was felt that this was a non-malignant ulcer, and there was nothing to suggest this being a tuberculous ulcer. A small piece was taken from the edge of the perforation for histological examination. The perforation was sewed with fine chromic catgut. The serosa of the cæcum was sutured over the closure with linen. The abdomen was then closed in the usual manner and two cigarette wicks were placed to the right iliac fossa.

Patient made an uneventful convalescence and she was discharged well on April 22, 1929. The operative wound was well healed throughout.

Pathological report. Source of specimen: appendix, biopsy of inflammatory mass in cæcum. Specimen consists of a roughly pyramidal piece of tissue 1.2 centimeters in height and 5 millimeters broad at the base. The lower 4 millimeters is yellowish-white and moderately firm, and seems to be fat. The remainder is uniformly softer and pink. Two sides of the pyramid are smooth and appear as if cut with a knife. The other two sides are granular and the angle between them is blunt. At the apex of the pyramid is a tiny notch. Accompanying the specimen is an appendix which measures 7 centimeters in length. The proximal 5 centimeters is 0.5 centimeter in diameter and soft to palpation. The distal 2 centimeters is represented by a thin fibrous cord 1.5 millimeters in diameter. The serosal surface is rough everywhere, but shows no exudate. When sectioned the lumen of the proximal part is found to be patent and to contain a small amount of bloody material. The distal lumen cannot be entered. The wall of the proximal part is slightly thickened, especially near the line of excision.

Microscopic section of the tissue from the edge of the ulcer shows the base of the pyramidal-shaped tissue to be composed of fat, the fibrous tissue septa of which are infiltrated with inflammatory cells, a large proportion of which are lymphocytes. There are also a few eosinophiles. Several broad septa are markedly œdematous. As the depths of this fat layer are approached, this cellular infiltration becomes more marked and here a few polymorphonuclears are intermingled with the other cells. The remainder of the tissue extending from the fat to the tip of the pyramid is more or less alike, and consists of very loose œdematous fibrous tissue, containing very few fibroblasts and a fair number of young capillaries. It is very heavily infiltrated with polymorphonuclears and some lymphocytes. There is also a moderate number of eosinophiles here, and a few endothelial leucocytes. Many of these cells are necrotic and have pyknotic nuclei, and there is considerable nuclear débris sprinkled throughout. Several spots show complete necrosis and take a uniform granular acidophilic stain. There is no indication of the original layers of the cæcum, except that near the tip of the pyramidal piece, there are again some fat cells embedded in the inflammatory tissue, and here are a few arterioles, and this might be taken to be the remains of the submucosa. There are no epithelial cells recognizable anywhere and no evidence of carcinoma. No amœbæ are found after careful search, and there is no evidence of tuberculosis anywhere. There are a few small areas of fresh hæmorrhage, but no old changed hæmoglobin to indicate previous hæmorrhage.

Section of the appendix shows the distal tip to be composed of a mixture of smooth muscle and fibrous tissue with only small knots of lymphocytes. No mucosa is seen anywhere. Proximal to this, the muscularis is thin and fibrosed for a considerable distance, although the mucosa is of good thickness. The mucosa here shows considerable eosinophilic infiltration, and scattered areas of lymphocytic infiltration are found in the muscularis and serosa. Diagnosis: chronic inflammatory tissue from cæcum; chronic obliterative appendicitis.

I felt it would be of considerable interest to ascertain whether there was any interference with the motility of the cæcum as a result of the operation or any gross evidence of pathology in the colon that might be revealed by X-ray studies. Therefore, following discharge from the hospital the patient was advised to have gastro-intestinal studies. The following is the report:

May 13, 1929. There was no obstruction to the passage of the barium enema. The base of the cæcum was slightly dilated. There were no filling defects but a moderate redundant sigmoid.

May 22, 1929. The œsophagus was normal. The stomach occupied a medium position. It appeared to be definitely narrowed near the pyloric end of the stomach, but this narrowing is probably due to spasm. The first portion of the duodenum was large but filled normally. At the 6 hour examination the head of the meal was in the distal portion of the transverse colon (normal motility). At 24 hours the large bowel almost completely emptied. Plain films of the gall-bladder region showed a slight suggestion of the outline of the gall bladder but no evidence of stones. Plain film of the urinary

tract showed some definite hypertrophic changes in the lumbar spine. There was nothing abnormal noted in the genito-urinary tract.

SUMMARY

A case of a simple, non-specific ulcer of the colon which perforated is reported. The gross and histological characteristics of this ulcer seem to be analagous to that of peptic ulcer, and therefore the question arises as to whether an ulcer occurring on the colon is not a local manifestation of the same general disease that peptic ulcer is. There is nothing in the past or present history of this patient that would suggest the cause to be mechanical, such as a decubital ulcer due to constipation. When the ulcer occurs on the right half of the colon it is difficult to differentiate this lesion from appendicitis. However, since in the vast majority of cases both conditions require immediate operation it is important to remember that when the appendix appears more or less normal or does not give evidence of a sufficient inflammation to produce the clinical picture, the cæcum and the ascending colon should be explored for the possibility of an ulcer.

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CLINICAL SURGERY

FROM THE HESSISCHE HEBAMMENLEHRANSTALT, MAINZ

THE THEORY AND PRACTICE OF INTRA-UTERINE CHARCOAL TREATMENT IN GYNECOLOGY AND MIDWIFERY

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THE favorable results reported by Benthin, of Koenigsberg, and Geller, of Breslau, led us to employ charcoal intra-uterinely in gynecological and obstetrical cases, and we have been able to obtain the same favorable results as reported by these authors.

Before responding to an invitation to test and recommend a new preparation or treatment—especially when good therapeutic precedures are already in existence—one must be convinced of the value of the new method and of its freedom from danger. In particular, one must understand the effect of the preparation—in the present instance, charcoal—and must then consider whether the new method of treatment (intra-uterine charcoal treatment) offers any great advantages over methods already available.

For the better understanding of intra-uterine charcoal treatment, I should like to preface my remarks with some brief observations on the importance and advantages of charcoal as a therapeutic agent.

The use of charcoal in medicine is probably not uncommon. Even in olden times, wounds and internal affections were treated with adsorbent substances. The most important quality of medicinal charcoal is, because of its porous structure and consequent great surface energy, its high power of adsorption, i.e., of fixing other substances. To this property charcoal owes its great use. The adsorptive capacity is manifested either in the adsorption of wound secretions or in the adsorption of bacteria and their toxins. The latter is of very great importance.

To gain a clear conception of the processes to which the name of adsorption phenomena is applied, it is necessary to have recourse to the department of physical chemistry, especially of colloid chemistry, in which the phenomena of surface energy are of great importance. I would refer in this connection to the paper[1] by Franz Koenig on "The Importance of Medicinal Charcoal in Therapeutics, and the Nature of Adsorption." In this paper a short sketch is given of the conception and nature of adsorption, and also of the practical employment of charcoal in medicine, i.e., of adsorption therapy, the range of indications of which is very extensive. For us as medical practitioners, these pharmaceutical details are of infinite value in enabling us to obtain for ourselves a satisfactory explanation of the mode of action of the application of charcoal. Adsorption depends upon "(1) the extent of the surface energy, i.e., the size of the free surface; (2) the surface tension between adsorbent (charcoal) and liquid; and (3) the surface activity of the liquid."

While, as a result of the adsorption of wound secretion, it is made much more difficult for bacteria to live on the drained tissue surface, it is true that, as a result of the adsorbent-fixative action on bacteria and on the poisonous properties of bacterial toxins, absorption into the blood stream is very greatly retarded, if not rendered altogether impossible. Weidmann has shown that charcoal powder has an adsorbent action even on bacteria in the blood. It is absolutely necessary, therefore, in cases of poisoning for example, that the quantity of charcoal should be sufficient to reduce the quantity of poison present to below the lethal dose. Van Amstel has demonstrated that the maximum dose of charcoal to be administered at once in man is 40 to 50 grams.

In addition to the great adsorptive power just described, the astringent action of charcoal plays a not inconsiderable part.

Is the local application of charcoal likely, it may be asked, to produce any injurious or detrimental effects? All the publications on the subject show beyond doubt that this is not the case. Geller and others have proved that no harmful action on the cells appears as the result of the application of charcoal, but that, in virtue of the property already mentioned, of abstracting fluid, a stimulant

[1] Pharm. Ztg., 1928, No. 101.

action is actually exerted on the tissues, which leads to hyperæmia.

The very fact that charcoal possesses such great therapeutic powers makes necessary especial care in selecting preparations of charcoal for medicinal use; such preparations must be of high standard. It is well known that the ordinary medicinal charcoal preparations on the market show very great differences in adsorptive capacity. The suitability of charcoal preparations for medicinal use is dependent not on the source (animal or vegetable) but solely on the method of treatment of the charcoal.

Up to the present, charcoal and, consequently, adsorption therapy have been used only in the treatment of the gastro-intestinal canal diseases such as acute poisoning with metallic salts, poisoning with organic poisons, poisoning produced by meat, fish, sausages, preserved foods; infectious diseases (cholera, dysentery, etc.) and intestinal auto-intoxication; hyperacidity and gastric ulcer (of special value in the latter on account of the acid-binding and ferment-inhibiting action of charcoal); excessive gas-formation (postoperative intestinal paresis); and the presence of large numbers of bacteria. Recently, however, charcoal therapy has been used, by means of intra-uterine application, in gynecology and obstetrics. Favorable results following the intra-uterine application of charcoal in metritis were first reported in veterinary medicine.

In what conditions in human gynecology and obstetrics is the charcoal treatment indicated? In abortion, especially febrile abortion, in puerperal endometritis, and in cæsarean section after rupture of the membranes. The conservative treatment of septic abortion first inaugurated by Winter, of Koenigsberg, is now generally recognized as correct.

The process of recovery is hastened by the intra-uterine application of charcoal by reason of the preventive and curative action of the charcoal. The determining factor in the success of the treatment is its application at the earliest possible moment, provided the process is still substantially localized in the uterine cavity and provided sufficient charcoal, in the form of granulated charcoal pencils, which, from the colloid-chemical point of view, approximate most closely the ideal powder, is introduced into the cavity of the uterus (insufflation of powdered charcoal is dangerous and therefore unsuitable for use in this treatment). The task of the charcoal thus introduced is to prevent, or at the least render difficult, the occurrence of any absorption from the residual fluid of the uterine cavity, heavily charged with bacteria and toxins, into the tissues or the blood and lymph apparatus. Such absorption, or the entry of the poisonous substances into the cells, is prevented or hindered by a protective layer, difficult of penetration, which is formed by the dissolved particles of carbon.

It is necessary to use a quantity of charcoal sufficient, after expansion, to fill the whole cavity of the uterus and to cover the decidua completely. Further bacterial invasion is thereby made impossible.

As already mentioned, it has been demonstrated by Geller that no injury to healthy, regenerated mucous membrane cells occurs as the result of too abundant application of charcoal in the case of a local focus in the uterine cavity.

The germicidal action is, indeed, heightened by the fact that, in consequence of the abstraction of water from the tissues, a stimulant effect, leading to hyperæmia, is simultaneously exerted on the healthy tissues. From these considerations and collected experiences, it is sufficiently evident that intra-uterine charcoal treatment offers great therapeutic advantages over the measures hitherto generally employed.

With uterine irrigations, a method now only rarely used, we obtain at most a temporary diminution of bacteria. The irrigating fluid is immediately evacuated, partly through the return-flow catheter, partly through the cervical canal. Its action on the foci of infection is, therefore, of short duration. There is, moreover, the danger of injury to the mucous membranes by the rigid catheter. Such injury makes a place of lessened resistance, sufficient to enable bacteria to break through the leucocytic defense. A further danger is that the bacteria-infested contents of the uterus may be washed upward into the fallopian tubes and possibly into the abdominal cavity.

Swabbing of the uterine cavity with ether and iodine, intravenous infusion of dextrose, and tamponage of the infected uterine cavity remain to be mentioned. The latter is supposed to act by absorption and at the same time serve as a means of escape for the uterine contents. Attempts have also been made to fight bacterial infection of the uterus by means of intravenous injections of trypaflavin, argochrome and collargol solutions, etc. Irradiation of the infected and metritic uterus with X-rays has likewise been employed therapeutically. All these methods of treatment are so variable in their results—while some are not wholly free from danger—that none of them can be called fully effective. All have their good and bad points. These factors of danger and variable results do not exist with charcoal therapy.

For charcoal therapy we make use of the pencils of granulated charcoal, measuring 3 and 5 centimeters in length and about 4 to 5 millimeters in thickness, which have been placed at our disposal by E. Merck, of Darmstadt. These pencils, which are prepared from pure, compressed granulated charcoal—kaolin is unsuitable on account of its forming lumps—are very convenient in form but are very easily broken. Gentle manipulation is necessary, as indeed is the case in all intra-uterine procedures. The fragility of the pencils has at the same time, however, the advantage that the least attempt, during their insertion, to overcome any resistance (narrow cervical canal, acute-angled anteflexion) by the use of force is met by the immediate breaking of the pencil, so that we are, as it were, debarred from causing injury.

What happens to the pencils in the cavity of the uterus? In the bacteria and toxin laden residual fluid of the uterus they begin to effervesce and to dissolve, at the same time exerting their therapeutic effect (inhibiting bacterial growth). They find themselves in a medium in which their disintegration is complete. That disintegration may, in isolated instances, be incomplete is shown by the fact that fairly large particles of charcoal are sometimes expelled again. Even after the lapse of 10 to 12 days we have found small remnants of charcoal in the posterior vault of the vagina. This fact has no influence on the favorable effects of the treatment. I shall again refer to these processes. The solution of the pencil can be facilitated by dipping it two or three times in distilled water before inserting it. By this means the outer parts of the charcoal pencil are softened and more rapid intermingling of the charcoal with the uterine contents is obtained. The insertion of the moistened pencils demands particularly delicate manipulation, as they are very much more fragile; on the other hand, by reason of the softened outer covering of charcoal, the possibility of injury and lesions of the mucous membrane is nil from the very beginning.

The processes which attend the interaction between the disintegrating charcoal pencil and the infected uterine fluid can be illustrated by preparing a fluid chemically similar to that contained in the uterus and treating it with the charcoal pencil in a vessel of some sort. To the accompaniment of effervescence and decrepitation, a thick, black pasty mass is produced, in which some solid particles of charcoal may remain undissolved.

In these experiments *in vitro*, which exhibit to us in some measure the intra-uterine processes, there is missing, however, the action on the charcoal pencil of the uterine contractions, which are of great value in effecting the solution of the charcoal and its union with the bacteria-infected contents of the uterus.

INDICATIONS

In the remainder of this paper I wish to speak of the range of indications for charcoal therapy and of the technique practiced by us.

Our treatment of abortion is strictly conservative, provided hæmorrhages do not call for our interference. We regard every febrile abortion as intentional, produced by external interference. The same may be held true of afebrile cases, only here the operator has proceeded with rather more cleanliness.

In numerous trials we have been able to prove experimentally that the granulated charcoal, introduced into the poison-containing media (before any toxic action on the tissue appears), prevents their extension and continuance. We speak of a "direct prophylactic action," by means of which many severe affections, and even death, can be avoided. The possibilities of this direct prophylactic action led us to insert one or two charcoal pencils, also, in every case of fever-free abortion which for any reason had to be actively terminated, always on the supposition that a local infection might be present. This prophylaxis is of still greater and more effective value in those cases which are admitted with temperatures of up to 38 degrees C. (100.4 degrees F.) and which it is necessary for us to terminate. We found in almost all cases that the temperature fell to normal in at most 24 hours and, what is worthy of note, that convalescence was uninterrupted and entirely free from fever, so that it could be assumed that the bacteria in the uterine cavity had been killed. In cases of febrile abortion, there is often during convalescence a subfebrile temperature extending over several days, and indicating in most cases an endometritis. The result of charcoal therapy is to eliminate this temperature and to secure a convalescence free from fever, and its success in this respect is the greater in proportion as the treatment is commenced early, i.e., before toxic damage to the tissues has set in.

There is a further field in which this direct prophylactic intra-uterine application of charcoal is indicated—cases of abortion which are admitted to the hospital with high fever and severe local infection. The possibility of securing better results in treating these cases by means of charcoal, is also mentioned by Benthin, who suggests that the infected uterine contents be rendered germ-free, or bacterial growth prevented at least, by inserting charcoal pencils as a first measure, *before* the infected case is actively terminated. In

similar cases we have begun by inserting charcoal pencils, and have been able in 24 hours at most to see whether the desired effect has set in, as evidenced by fall of temperature, improvement in the general condition, and almost immediate cessation of any malodorous discharge that may be present. A critical examination of each individual case must naturally precede treatment, for we can expect to influence the bacteria by means of intra-uterine charcoal therapy only when the pathological process is strictly localized in the uterine cavity. After this direct prophylactic preliminary treatment, we can be sure of having a uterine cavity which is in some measure free from bacteria, so that less danger is involved in subsequent active procedures. Cases treated in this way have confirmed in practice our theoretical conclusions. The further course was in all cases free from fever, the fall in temperature critical. In these cases a part is played not only by the adsorbent and astringent action of the charcoal, but also by the charcoal pencil as a foreign body. The uterus reacts with contractions, so that placental remnants which have become purulent are partly detached, if not entirely expelled. As soon as detachment occurs, the dissolved charcoal can immediately exert its therapeutic powers at the site of adhesion, and so prevent further ascent of the bacteria.

Puerperal metritis is also very favorably influenced by intra-uterine charcoal therapy, so that we now include this condition among our indications for the treatment. As soon as evil-smelling discharge and subfebrile temperatures set in, the patients are treated for 5 to 6 days with ergotin or gravitol (either 20 drops thrice daily or, better, a double dose of one of these medicaments on each of 3 successive days) and with the ice-bag; the head of the bed is also raised 25 centimeters, so as to facilitate escape of the discharge. If no improvement is obtained by these means, we introduce charcoal into the uterus on the seventh day, under the strictest aseptic precautions. In all cases a reduction in temperature was obtained on the next day, and on the second day at latest the temperature fell to normal. The fetid odor disappeared at once. In over 90 per cent of the cases it was unnecessary to insert charcoal pencils more than once, a fact which says much for this method of treatment. In several cases of pyometra we were able to bring about complete recovery, with lytic fall of temperature, only after several applications of charcoal. Even in these cases, however, the evil lochial odor—often very unpleasant both to the patient and her neighbors—disappeared immediately after the very first application, a result which has a considerable influence in improving the patient's general condition.

As a result of our experience, I should like to extend the range of indications still further and include therein a class of cases often complicated by prolonged wound suppuration. These are cases of cæsarean section after rupture of the membranes. We cannot know how far an infection of the uterine cavity has taken place. Our statistics of cæsarean section for the ten years from 1918 to 1928 showed one abdominal abscess in cases of operation before rupture of the membranes, 10 abdominal abscesses in cases of operation after rupture of the membranes, and almost always an endometritis with fever, which in most cases lasted over a week.

Accordingly, in such cases we insert several (3 to 5) granulated charcoal pencils into the uterine cavity after expulsion of the placenta. It is here quite impossible to cause even the slightest injury, as the uterine cavity lies open. I regard this method as the safest way of obtaining local prophylaxis of the uterine cavity. In swabbing with ether, tincture of iodine, or dry swabs there is the great danger of transferring bacteria from one part to another.

Prophylaxis against an infection which is still invisible is also, in any case, a less severe and less dangerous proceeding than the treatment of an evident infection of the endometrium. From this therapy we have as yet seen no disadvantages, but only advantages.

We, therefore, employ charcoal pencils

1. In cases of infected abortion, before or after clearing out, according to the condition of the patient;

2. In cases of puerperal endometritis—not before the seventh day of the puerperium;

3. Prophylactically, in cases of cæsarean section after rupture of the membranes.

TECHNIQUE

The technique of intra-uterine charcoal therapy is very simple. The necessary instruments (2 specula, 1 hooked forceps, 1 bullet forceps, and 1 long tweezers) are previously boiled; before insertion of the specula, the orifice of the vagina is cleansed and then the vagina washed out with aluminium acetate. The portio vaginalis is exposed and the anterior lip of the os uteri hooked. The external os uteri is cleansed from mucus and blood. The charcoal pencil is grasped at one end with a dressing-forceps, dipped two or three times in sterile water, and then carefully inserted through the cervical canal to beyond the internal os uteri. The cervical canal is almost always so

wide open that the charcoal pencil can be introduced without difficulty. We do not agree with Benthin in his recommendation of dilatation when the os uteri is occluded. According to the severity of the case and the size of the uterine cavity, two or three pencils may be introduced, and then distributed in the cavity, the first more toward the right, the second more toward the left tubal angle, and a third if necessary in the middle. Generally, however, one pencil of 3 centimeters in length is sufficient. After removal of the dressing forceps, one hears the crackling and effervescence already mentioned as taking place when the charcoal pencil fixes the uterine contents. The crushing and mixing of the charcoal are greatly assisted by the muscular contractions which, as previously noted, are produced by the charcoal pencil in its capacity as a foreign body. In order to prevent immediate escape of the charcoal paste or foam through the cervical canal, a thin strip of gauze is placed in the cervix, with one end projecting from the vaginal orifice. After the lapse of 3 to 4 hours the gauze is withdrawn, as it can then be assumed with safety that intermixture of the charcoal and the uterine contents is complete, and that the walls of the uterine cavity have become covered with the charcoal suspension. As I have already mentioned, comparatively large particles of charcoal are found in some cases in the posterior vaginal vault. The undissolved portions of charcoal are expelled by the uterine contractions. The fact that fairly large particles of charcoal may remain undissolved is capable of explanation as follows: (1) there may be an excess of charcoal in proportion to the size of the uterine cavity, i.e., the uterine contents are saturated; (2) some parts of the charcoal pencil may be too strongly compressed or may be lying more in the cervical portion of the uterus, and so cannot be completely dissolved by the bacteria-containing residual fluid. An excess in the amount of charcoal introduced is never to the disadvantage of the treatment, as one can then be sure that the whole uterine cavity is uniformly filled, i.e., is lined with a coating of charcoal capable of exerting an adequate adsorptive action. The thicker the wall represented by this coating, the greater will be its fixing effect.

To the details already given I would add that those of our cases which come within the indications mentioned have not simply been subjected to treatment with charcoal alone. In many cases, naturally, our previous methods of treatment have had to give way to charcoal therapy, to enable us to get an accurate view of the effects of the application of charcoal alone. The cases thus treated almost all led to the desired result. We have, indeed, obtained the impression that we considerably shortened convalescence without detriment to the premanence of the recovery. When we were in position to judge the results of charcoal treatment alone, we assisted the process of healing by means of ergotin or gravitol and the ice-bag. The decisive factor in bringing about the rapid and favorable results, however, was the charcoal alone.

Unlike Benthin, we have not strictly limited our range of indications, but have tried to obtain conclusive proof, in as wide a variety of cases as possible, of the great therapeutic powers lying dormant in charcoal. Our observations give us ground for placing our favorable results on a level, in every respect, with those of Benthin and Geller. These authors conclude their discussion with an appeal for further testing of the method. I should not like to close my paper without repeating this appeal, for critical consideration of the results obtained shows the treatment to be worthy of trial. Everything new, however, is met with a certain skepticism. "Warum denn in die Ferne schweifen, wenn das Gute liegt so nah!"[1] Why should I still further overload a sick body with high molecular intravenous solutions; why should I wash or swab out the cavity of the uterus, and thereby run the risk of producing injuries or of facilitating a further ascent of the pathological process? The reported results of the charcoal treatment, both in human and in veterinary medicine, are so favorable that they cannot be rejected at once with a decisive "No." The intra-uterine application of charcoal is completely free from danger, the medicament is very cheap, and the period of illness is greatly shortened. The symptoms are instantaneously influenced, never for the worse. The body responds with an immediate improvement in the patient's general condition and in the local process, with a fall in temperature and in the pulse rate.

INTRAVENOUS CHARCOAL THERAPY

What I have said so far relates only to local—intra-uterine—charcoal treatment. I should like now, within the framework of this paper, to touch upon the subject of intravenous charcoal therapy. This first came into use, simultaneously with the intra-uterine treatment, in veterinary medicine.

A suspension of charcoal was injected intravenously in septic affections. This intravenous application of charcoal was first adopted in human

[1] "Why, then, go abroad seeking afar, when the good lies close at hand!"

medicine—and with very good results—by Wedekind, at the I. Medical Clinic University of Cologne (Prof. Kuelbs), for the purpose of actively attacking tuberculosis. In his summary, Wedekind says: "By the intravenous administration of very small amounts of charcoal dust, even severe exudative and proliferative forms of human pulmonary tuberculosis can be brought in a short time to induration. A pre-requisite for this form of therapy is that too great portions of the lungs should not already have undergone ulcerative disintegration, and that the body should still be capable of reaction. Relatively recent processes are most capable of recovery.

"No aggravations attributable to the treatment were ever manifest. Symptoms of intoxication did not appear. This influence of charcoal dust on the course of tuberculosis is explained by the activation of the reticulo-endothelial defensive apparatus which it produces, and in particular by the mobilization of the connective tissue histocytes of the lungs."

Another very interesting anatomico-physiological study of the action of intravenous charcoal therapy in pulmonary tuberculosis is one by H. Gickler, which appeared in the *Beitraege zur Klinik der Tuberkulose.*

I have purposely referred to these papers in some detail, because in part they provided us with factors for comparison with the observations made by us in the intravenous application of charcoal in sepsis, and in part gave us an explanation of processes which are exceedingly important in the problem of the treatment of sepsis. As I have here touched on the subject of sepsis, I should like to mention the paper published by Louros and Scheyer on "streptococcal infections, the reticulo-endothelial system, their relations and their amenability to therapy." The two authors sum up their very interesting observations as follows: "From the investigations described it appears that in animal experiments it is possible, by means of injections of charcoal to exert a favorable influence on the outcome of a streptococcal affection, in one-third of the cases, this result being attained by way of a general improvement in the functioning of the reticulo-endothelial system, and in particular by a heightening of the phagocytic activity of the reticulo-endothelial system, especially toward introduced bacteria. The charcoal acts first by altering the hydrogen-ion concentration of the media in the direction of an acidity which checks bacterial growth (Dresel). The charcoal then exerts its chief action as an adsorbent and serves as a vehicle for the bacteria, to the phagocytosis of which it stimulates mechanically the reticulo-endothelial system. The action is to be regarded as purely non-specific, and appears to be particularly favorable by reason of the fact that the mechanical stimulant effect on the reticulo-endothelial system is combined with the physical phenomenon of adsorption and with the physico-chemical phenomenon of alteration of the hydrogen-ion concentration."

So far as our experiments and experiences, up to the present, in the intravenous charcoal treatment of sepsis allow of comparison, we can say that much valuable investigation can still be done in this department. At all events, the cases of sepsis which we have so far treated in this way have provided us with a plan of work, for the carrying out of which a large number of interesting experiments of very varied nature are necessary, some of these representing in themselves a complete field of work. We shall report on these matters elsewhere.

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FROM THE SURGICAL CLINIC, STATE UNIVERSITY AT KASAN

LOCAL ANÆSTHESIA IN ABDOMINAL SURGERY[1]

PROFESSOR ALEXANDER W. WISHNJEWSKY, KASAN, U. S. S. R.
Director of the Surgical Clinic

I BELIEVE that the ideal anæsthesia in surgery will be that which will cause the patient's organism no insult outside of the operative field. Local infiltration anæsthesia comes closest to this ideal. However, the classical infiltration anæsthesia of Reclus with the concentrated solutions of cocaine could not be accepted as harmless for the organism. Work with concentrated solutions is based upon the principle of osmosis and diffusion and in this respect the solutions are not constant in their effect. This fact has contributed greatly toward the disrepute in which infiltration anæsthesia has fallen and even the brilliant investigations of Braun, who introduced novocain and adrenalin in local anæsthesia, have helped little to make popular such means of inducing anæsthesia.

My method of inducing local anæsthesia consists in the injecting, layer by layer, of large quantities of a ¼ per cent solution of novocain in Ringer's solution from which bicarbonate of soda is removed, with the addition of 2 minims of adrenalin to each 100 cubic centimeters of solution. Large amounts of solution are used for injection into the various anatomical layers of the operative field; as a result, large serpentine anastomosing infiltrations are formed, which block the nerves by direct contact and pressure rather than by diffusion. Occasionally the solution is injected slightly away from the immediate operative field, but always close to it. The tissues may be incised immediately upon injection without the need of waiting for "diffusion" of the drug.

No single one of all the various methods of local anæsthesia is applicable to all types of surgical operations within the abdominal cavity. It is true that local anæsthesia is frequently used in performing complicated surgical procedures in the peritoneal cavity. However, there is no single, well systematized method of inducing local anæsthesia that may be used in all major operations within the abdomen. Mikulicz and Schleich introduced the method of using infiltration anæsthesia for the abdominal wall, but after the peritoneal cavity was opened they continued the operation without further anæsthesia. Finsterer, Farr, and others suggested that mesentery and ligaments which are to be handled during the operation be anæsthetized by means of injections of novocain. Laewen and Siegel worked out the technique of paravertebral anæsthesia, while Kappis and Braun that for splanchnic anæsthesia. The latter method has become popular although nitrous oxide is used with it.

I shall describe here the method of local anæsthesia which is being used in my clinic in abdominal surgery. My method is a further development of a combination of infiltration anæsthesia and mesenteric anæsthesia. That local anæsthesia by injection into the mesentery as suggested by Farr and Finisterer is far from being satisfactory, is easily seen from the difficulties usually encountered in its use. I have witnessed situations in which the surgeon is lost in an effort to locate a point for the injection of the novocain into the mesentery, and each time the needle was inserted the surgeon ran the risk of puncturing a blood vessel. Years ago I had the same difficulties, but of course, with growing experience, I have learned to master such situations. However, I have been struck by the fact that the technique used for this type of anæsthesia is purely casual in character and that the method of procedure is without system or definite plan. The results of my surgical work changed radically after I worked out systematically the details of producing local anæsthesia, and I wish to describe here the technique I have worked out in producing anæsthesia in relation to the various important abdominal organs.

TECHNIQUE

The stomach. I shall consider here the method of producing anæsthesia in a patient requiring resection of the stomach. Such an operation is a supreme test not only because of the difficulty of the operation itself but because of the condition of the patient.

1. The midline along the proposed line of incision is infiltrated with novocain after which the subcutaneous layer is infiltrated. Infiltration must be sufficient to cause a true swelling of about two fingers' breadth, since only then will the anæsthesia be effective for a period of about 3 hours. The incision is made and the midline dissected (Fig. 1).

[1] Translated from the Russian by Dr. Anatole Kolodny.

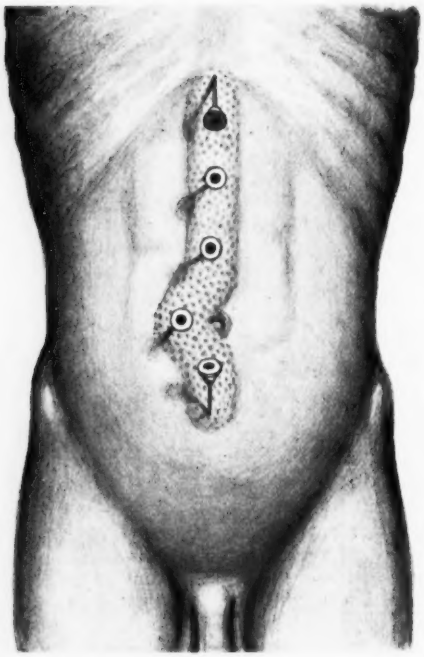

Fig. 1.

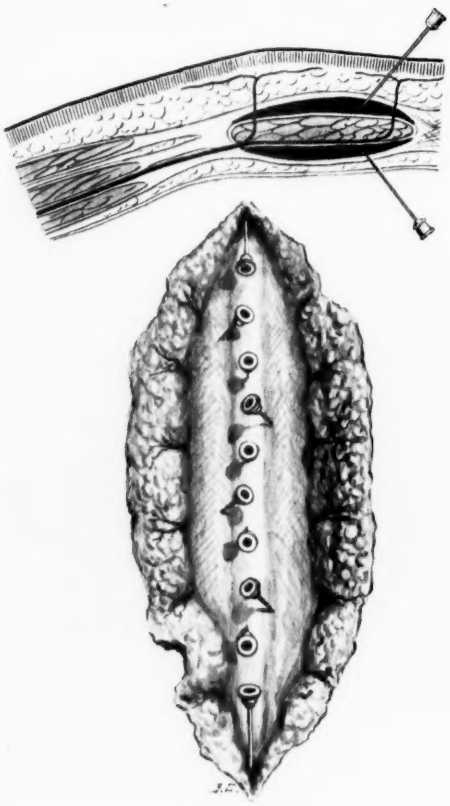

Fig. 2.

2. The midline is then injected in such a way as to infiltrate the preperitoneal fat. It is essential to direct the needle under the anterior sheath of the rectus muscle on either side of the midline.

3. After the incision of the midline the parietal peritoneum is anæsthetized. The preperitoneal tissues are retracted and novocain is injected about the parietal peritoneum and especially into the posterior sheaths of the recti on either side (Fig. 2).

4. The peritoneum is incised and the transverse colon is delivered into the wound, turned up, and the inferior surface of its mesentery exposed. An avascular point close to the root of the mesentery is chosen for the first injection. The subsequent injections are so made into the borders of the area which has become œdematous from the first injection that on one side the injected fluid spreads behind the posterior parietal peritoneum downward toward the root of the mesentery of the small intestines, and on the other side expands throughout the root of the mesocolon thus separating both leaves of the peritoneum and extending upward toward the peritoneal envelope (serosa) of the duodenum (Fig. 3). The spreading of the fluid toward the root of the mesentery of the small intestines makes it essential to keep the intestines out of the way; this point is not taken care of in the usual method of administering splanchnic anæsthesia. When the transverse colon is turned downward at this stage of the procedure, the œdema caused by the injection is easily seen; this œdema is shimmering through the superior peritoneal leaf of the mesocolon especially on the right side, where it is not covered by the gastrocolic ligament. Supplementary injections into the borders of these œdematous areas are easily made, and the danger of puncturing a blood vessel is avoided. As a result the duodenum is bathed, so to say, in the injected fluid which readily spreads beneath its serosa (Fig. 4). The mesentery of the stomach is then injected along its lesser curvature, in a direction toward the cardia. The gastrocolic ligament is divided and immediately the upper leaf of the mesocolon comes into view. Several more injections are made here, and the fluid

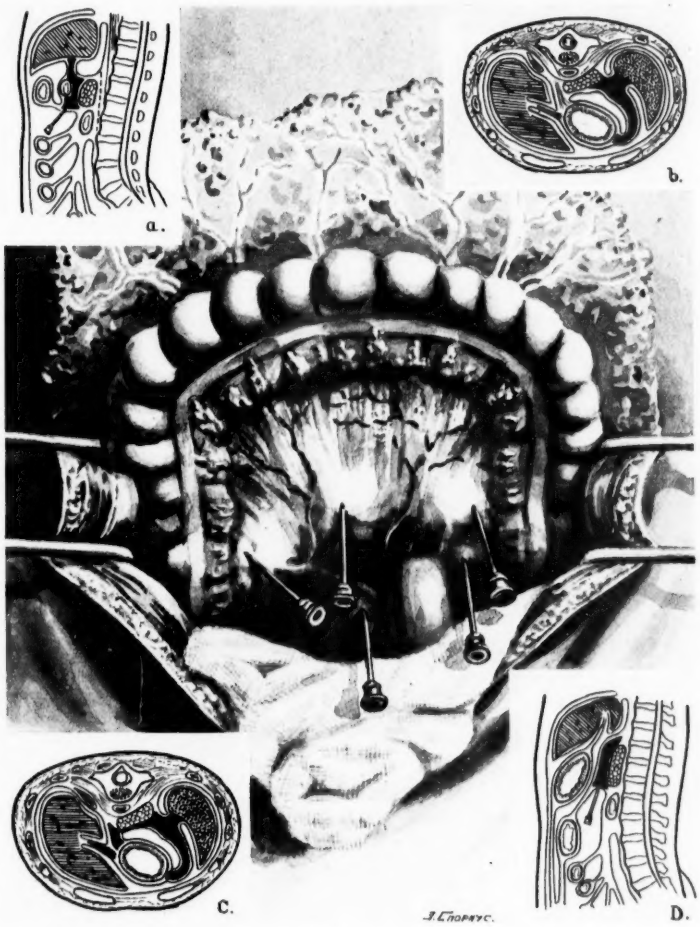

Fig. 3.

gradually expands toward the pancreas and appears behind the parietal peritoneum at the level of the lesser curvature. Finally the plica gastropancreatica and the gastrosplenic ligament are infiltrated. It is now possible to perform any variety of resection of the stomach (Fig. 5).

Bile ducts. The principles described underlie my method of administering local anæsthesia in surgery of the bile ducts. The novocain is so injected that infiltration is extensive and rapidly spreads along exact anatomical paths. An oblique subcostal incision is made after injection of the abdominal wall in layers. The parietal peritoneum is anæsthetized, the injection being started well beyond the line of incision. The transverse colon is delivered and the right half of its mesentery from the root to the periphery is injected. The colon is then turned forward and downward. By pulling down the hepatic flexure slightly, one can easily see between the leaves of the mesocolon the injected fluid spreading toward the posterior abdominal wall. One continuous injection is made into the margins of the œdematous area until the free edge of the hepatoduodenal ligament becomes infiltrated. Then the colon and omentum are replaced in the abdominal cavity, a large gauze pad directed toward the kidney is inserted to pack off the intestines, and the injection of the gall bladder proper is begun.

If the gall bladder has an anatomically intact

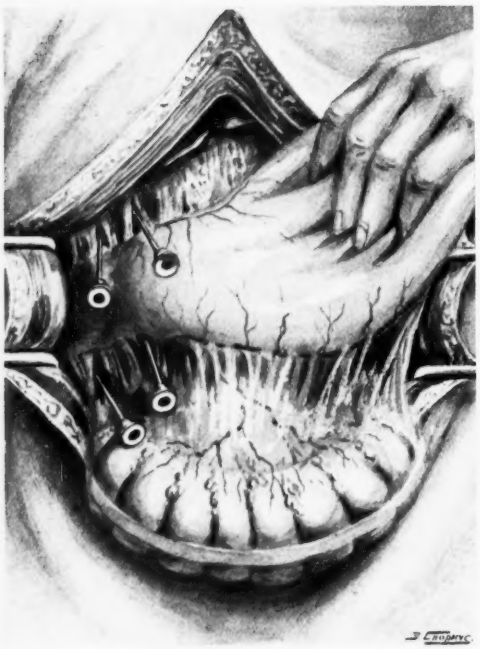

Fig. 4.

serosa, a subserous injection of the solution is made until the fluid is seen to have spread toward the ducts, and there to have joined the œdema caused by the previous injections of the hepatoduodenal ligament. The injected fluid easily separates the gall bladder from its liver bed. Such an injection contributes greatly to the ease with which a subserous cholecystectomy is accomplished. If the gall bladder is shrunken, scarred, or tensely packed with stones, one is forced to fall back upon scattered individual small point injections, even though the field of the ducts proper remains completely anæsthetized from the previous systematic serpentine injections (Fig. 6).

Spleen. A freely movable spleen without adhesions can be easily removed after the line of incision through the abdominal wall has been anæsthetized, but when the spleen is adherent it is necessary to administer a strictly systematic anæsthesia, not unlike that used for operations on the stomach and bile ducts. After the abdominal cavity is opened through a left oblique subcostal incision, the left half of the mesentery of the transverse colon is anæsthetized in the fashion outlined. A portion of the gastrocolic ligament just sufficient to allow approach to the upper leaf of the mesocolon is divided. After this section is thoroughly anæsthetized the gastrosplenic ligament on the left side is attacked in the same way. Occasionally one will have to inject separately adhesions between the spleen and the diaphragm. That this method is practicable I have been convinced since it has proved successful in my last ten consecutive splenectomies.

Large intestines. I know of no report in the literature of a complete removal of the large bowels under intra-abdominal local anæsthesia. I have used the principle of the serpentine injections in administering local anæsthesia in two patients in whom complete removal of the large intestines was considered indicated. The technique may be described as follows: After a midline incision is made, the transverse colon is delivered and its mesentery is injected as already outlined. The fluid is directed widely behind the posterior parietal peritoneum toward the root of the mesentery of the small intestines. This mesentery is then completely anæsthetized by means of additional injections. The small intestines are then delivered from the peritoneal cavity, retracted to the left, and the cæcum and ascending colon are exposed. The mesenteries of the cæcum and ascending colon are injected in the same systematic way. An incision in the peritoneum is then made near the root of the cæcum, and the removal of the bowel is commenced. This removal of the bowel is performed gradually in steps so that repair of the defect in the parietal peritoneum immediately follows upon the division of the mesentery in the given region. Before the splenic flexure is approached, the small intestines are turned to the right and the descending colon is anæsthetized in the usual manner.

In both patients a complete removal of the large intestines was done. Both stood the operation well and left the operating table with a pulse of 70 to 72. The postoperative course was free from complication with one exception—a transient physiological diarrhœa. Both patients were previously operated upon repeatedly for a diffuse ulcerative colitis. Attempts at shortcircuiting did not affect the patients' condition and they remained bedridden until complete colectomy was done. At present the patients are in perfect health. I am emphasizing the fact that the small intestines remain without the peritoneal cavity during the entire removal of the colon.

Pelvic organs. My method of producing local anæsthesia by means of large serpentine anastomosing infiltrations is perfectly applicable in gynecology. Here anæsthesia is begun by the formation of extensive infiltration along the an-

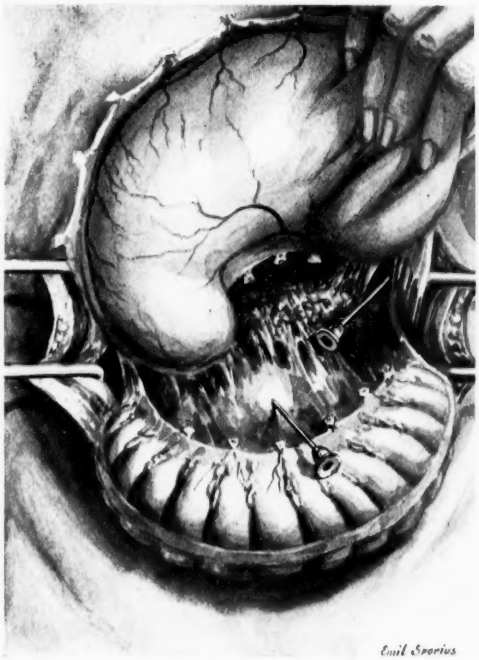

Fig. 5.

Fig. 6.

terior aspect of the sacrum. This infiltration is of no blocking importance in the sense of Braun's parasacral anæsthesia, when one has to seek each individual sacral foramen. This infiltration is caused merely for future anastomosis with the infiltrations made along the promontory and the innominate pelvic line. To produce presacral infiltration the injection is begun in the midline about 1 inch posterior to the anus. Immediately after the needle is inserted, the fluid is slowly injected, the needle being gradually moved toward the anterior aspect of the sacrum. The solution easily finds a line of cleavage through which it spreads in front of the sacrum. I inject here about 300 cubic centimeters of the novocain preparation. The anæsthesia of the abdominal wall, of the parietal peritoneum, and in case of adhesions, of a small portion of the adjoining intestinal mesentery, is done in the manner outlined. Occasionally, the round and broad ligaments may need supplementary injection.

SUMMARY AND CONCLUSIONS

It is apparent that my method of producing local anæsthesia consists in a blocking of the nerve plexus, the blocking being begun in the periphery and being gradually extended toward the centers. Of course, during the operation the anæsthetic may spread toward the ganglions but it never suddenly encroaches upon them. This fact contributes much to the physiological safety of the method. The method has all the advantages of splanchnic anæsthesia without any of its disadvantages. It is not aggressive, it is technically easy, it can be used in the upper abdomen through any abdominal incision and even in the obese patient is practicable, it requires no general anæsthesia, and it is not followed by a fall in the blood pressure. However, this method differs from direct intra-abdominal anæsthesia in which the anæsthesia is carried along separate spheres of action or along the mesenteric vessels, methods which reflect and depend upon the intuition and experience of the individual surgeon, but which lack exact systematic performance.

This method of local anæsthesia based upon the use of large amounts of solution of novocain for the production of serpentine anastomosing infiltrations along anatomical layers leads to an immediate blocking of all the nerves coming in

direct contact with the solution. One is not forced to postpone the incision, awaiting the "diffusion" of the solution to the nerve trunks.

Because of the weakness of the solution of novocain, because of the gradual injection during the entire surgical operation, and especially because of the immediate incision and spontaneous removal of the injected fluid, no danger of intoxication is seen in this method of inducing local anæsthesia. The systematic injection of the solution layer by layer, contributes greatly to the ease of orientation in the anatomical structure of the organs handled; as, for example, the dissection of the gall bladder from the surrounding structures, the freeing of the adherent appendix, etc.

The co-operation of the patient is, of course, a prerequisite in this method of anæsthesia, and therefore, the technique is contra-indicated in children and in semiconscious patients. Other contra-indications are suppurative conditions of the abdomen. In all other instances this method of inducing local anæsthesia has proved practicable and reliable during the years of its employment in my clinic.

THE OPERATIVE TREATMENT OF UNUNITED FRACTURE OF THE NECK OF THE FEMUR

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I HAVE read with interest and pleasure Dr. Albee's paper[1] on the treatment of ununited fracture of the neck of the femur, particularly because of his unqualified endorsement of the abduction method and because it is evident that he no longer favors the immediate open operation, still employed on various pretexts by certain surgeons. I agree with him that in cases of ununited fracture, in which the fragments retain a fairly normal contour, the autogenous bone graft operation, of which he is the leading exponent, offers the best assurance of union.

There remains then for discussion only the third and at the present time much more important class in which, because of destruction of bone, the restoration of an approximately normal relation is impracticable. In the treatment of this group, leaving out of consideration the palliative bifurcation operation of Lorenz, there are practically speaking but two alternatives: the reconstruction operation and the procedure employed by Dr. Albee.

The reconstruction operation was first performed in 1916 but was not described until 3 years later.[2] Its design was to provide a secure support in locomotion and to restore as far as possible normal muscular control. An improvised neck was constructed by utilizing the bearing surface obtained by removing the trochanter at its base, and leverage was restored by transplanting it with its attached muscles to the outer side of the femur at such tension as would assure security of the joint (Figs. 1 and 2).

In this paper the various procedures then at command were compared from the functional standpoint, including that of Dr. Albee which had been described in 1919 in his "Orthopedic and Reconstruction Surgery." From these diagrams it will appear that the trochanter was separated only sufficiently to permit the introduction of the upper extremity of the femur to the acetabulum (Fig. 4).

Its development to a physiological bone lever did not appear until 6 years later,[3] and a further modification is presented in his last paper (Figs. 5, 6, 7, and 8).

It may be noted that the figure of 1919 presents no indication of a design to increase leverage and shows a narrow and irregular articulating extremity, while that of 1929 is smooth and bulbous. It would seem that the functional results of the

[1]Albee, Fred H. Surg., Gynec. & Obst., 1929, xlix, 810.
[2]Whitman, Royal. Surg., Gynec. & Obst., 1921, xxxii, 479.
[3]J. Am. M. Ass., 1925, 85.

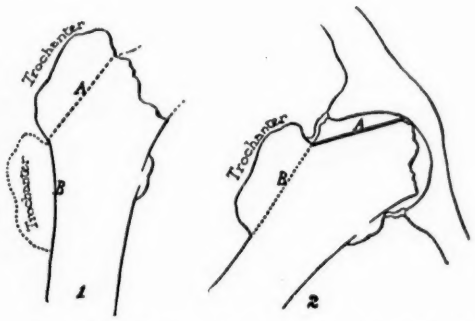


Fig. 1, left. The condition in ununited fracture of the neck of the femur in which the neck has entirely disappeared.
Fig. 2. The new bearing surface provided by removing the trochanter and transplanting it lower down on the shaft. (Figs. 1 and 2, from Surg., Gynec. & Obst., 1921, June.)

Fig. 3. A final result of the reconstruction operation.

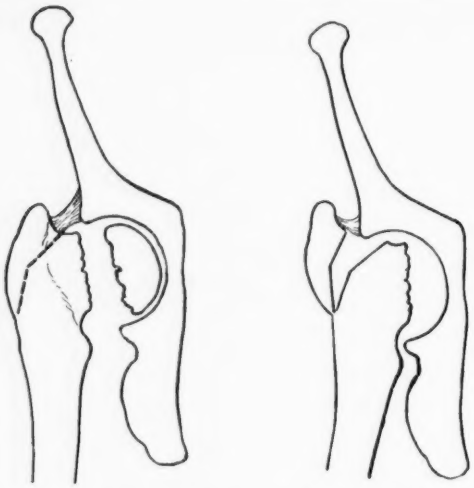


Fig. 4. "Old ununited fracture of the neck of the femur with erosion of most of head and neck and with marked osteoporosis of the remaining shell of the head. The trochanter is separated with osteotome, as shown by dotted lines in figure at left and is forced outward with the overlying soft structures below as a hinge. The removal of the head then allows the neck to be displaced into the acetabulum." (Albee, *Orthopedic and Reconstruction Surgery*, 1919.)

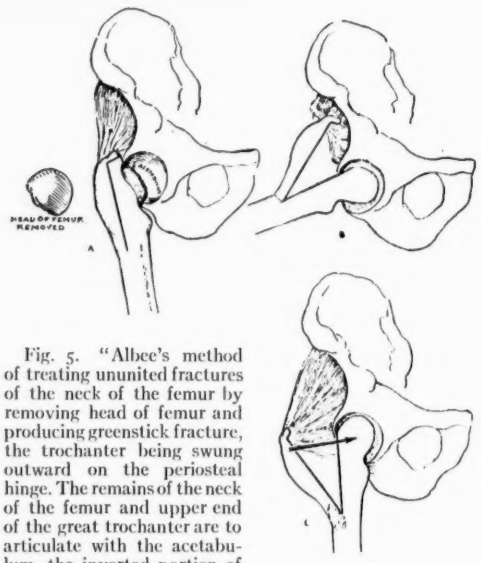


Fig. 5. "Albee's method of treating ununited fractures of the neck of the femur by removing head of femur and producing greenstick fracture, the trochanter being swung outward on the periosteal hinge. The remains of the neck of the femur and upper end of the great trochanter are to articulate with the acetabulum, the inverted portion of the capsular ligament contributing to the formation of the new articulation." (Albee J. Am. M. Ass., 1925.) As contrasted with the original this illustrates the evolution of the physiological lever.

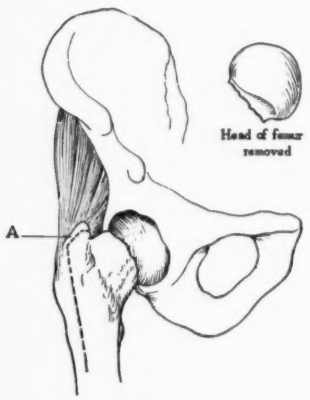


Fig. 6.

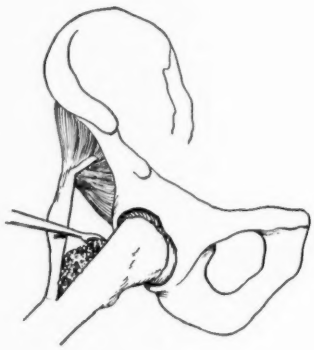


Fig. 7.

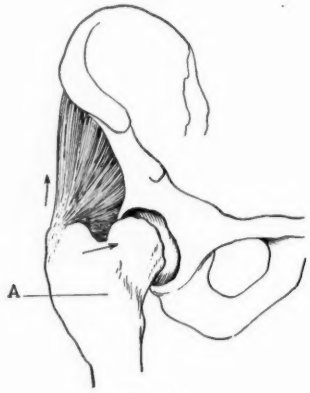


Fig. 8.

Fig. 6. "Schematic drawing of author's reconstruction operation with removal of femoral head; dotted line indicates bone section of upper end of femur by broad osteotome."

Fig. 7. "Displacement of upper end of bone muscle lever outward by abduction of hip which automatically thrusts the new femoral head into the acetabulum, at the same time holding the bone muscle lever in oblique relation to the shaft. The angle is then filled with cancellous bone material from greater trochanter as indicated."

Fig. 8. "Drawing from X-ray film showing consolidation of union of bone muscle lever with main portion of femur, angle being filled in at *A*." Albee, Surg., Gynec. & Obst., 1929.

operation as performed in 1929 must differ materially from those obtained in the earlier cases, but no distinction is made in the table of statistics.

Dr. Albee states that "his operation is the simplest yet devised," yet it entails stripping the muscles from the ilium over a large area and splitting off a wide section of bone from the femur. This is then forced outward leaving a triangular space approximately 4 by 3 inches to be filled by prospective bone formation.

The reconstruction operation by contrast has not been changed in any essential since its introduction. It has the advantage of simplicity of design, directness of approach, and definiteness of execution. There is no division of muscles, the anatomical adjustments are completed at the time of operation and are therefore independent of the nutritive processes on which Dr. Albee counts to assure the stability of the lever.

A final point at issue is that of nomenclature, since Dr. Albee claims the same name for his operation.

It seems to me that the reconstruction operation justifies its title not merely by priority but by design and accomplishment, while in both particulars Dr. Albee's operation presents a complete divergence from the natural construction. I suggest, therefore, in order to avoid confusion, that it be designated by one of its subsidiary titles preferably the "physiological bone lever operation," which will indicate the essentia! distinction between the alternative procedures.

Fig. 9. "Result after bone-muscle lever has united in place; illustrating the wide distance between the great trochanter and the side of the pelvis in proximity to the rim of the acetabulum, thus allowing for generous abduction." (Albee, 1925.) This illustration shows the actual condition far more clearly than the somewhat fanciful diagrams, particularly the shape and area of the bearing surface. Furthermore, that the displaced trochanter is relatively elevated, lessening the distance between the origin and attachment of the abductor muscles, and therefore their leverage. Contrast with Fig. 3.

THE USE OF IODIZED OIL (LIPIODOL AND IODIPIN) IN THE DIAGNOSIS OF JOINT LESIONS

PHILIP H. KREUSCHER, M.D., F.A.C.S., AND H. KELIKIAN, M.D., CHICAGO

THE value of iodized oil in the diagnostic visualization of bronchiectatic cavities and spinal canals is well recognized. It has occurred to us that because it is non-toxic, non-irritating, and shows a splendid shadow on the X-ray film, iodized oil should be suitable for a similar purpose in major joints. With this in mind, we have used it in several of the major joints, especially the knee and hip joints. Valuable information is obtained concerning the conformation of the joint cavity, its capacity, the communication with bursæ and with other diseased processes in the joint or communicating with it. Such pathological changes as hypertrophy of the synovial membrane, destruction or adhesions of the synovial capsule, erosions of the articular cartilage, and cavitations into adjacent bones may be well demonstrated by the use of iodized oil injections followed by immediate and subsequent roentgenographs.

It is interesting to note that in the various types of arthritis in which one would expect the joint cavities to be large and contain a large quantity of oil, they will contain only a small portion because of the partial obliteration of the synovial pouch. The more nearly normal the joint, the more evenly is the oil distributed. Patchy distribution indicates pathology. In one of our hypertrophic cases, 100 cubic centimeters was injected into the knee joint without producing undue distention or excessive pain (Fig. 9, Case 5).

The technique of the injection of the knee joint is very much the same as for injection of any other material. A point is selected on the outer side of the knee about 1 inch above the external lateral aspect of the patella. Through a small puncture opening in the skin the needle is inserted under the patella into the joint cavity. Any fluid which may be in the joint is completely aspirated with an aspirating syringe. The warm iodized oil is then injected until the capsule is completely distended. Whenever possible, the injection should be made without general anæsthesia. A roentgenogram is taken immediately after the injection. The films thus taken anteroposteriorly and laterally show the oil in the various recesses of the free joint cavity. After manip-

Fig. 1.

Fig. 2.

Fig. 1. Atrophic arthritis. Note the limited extent of joint capsule filling due to synovial adhesions. (Case 1, Mr. H. W.)

Fig. 2. Shows filling of joint in Case 1 after a second injection 3 months after first injection. The patient had been treated by immobilization, diathermy and amidoxyl benzoate injections.

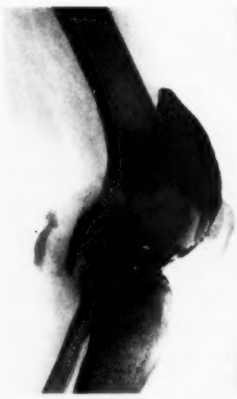


Fig. 3.

Fig. 4.

Fig. 3. Hypertrophic arthritis showing shadow of iodized oil in the joint cavity with the popliteal bursa partially filled, immediately after injection in Case 3.

Fig. 4. Film 15 minutes after injection and following flexion and extension exercises of the knee. Note complete filling of popliteal bursa, also point of exit of the bursa into the synovial pouch. Iodized oil shadow is limited by the intact synovial sac. (Case 3.)

Fig. 5.

Fig. 6.

Fig. 7.

Fig. 8.

Fig. 5. E. A., tuberculosis of knee joint, before injection, showing cavitations into both tuberosities of the tibia. (Case 4.)

Fig. 6. Film immediately after injection showing large popliteal compartment of the synovial sac and tongue-like projections of the oil into small cavity in tuberosity of the tibia. (Case 4.)

Fig. 7. Partially filled cavitations in the tibia. (Case 4.)

Fig. 8. Two weeks after injection. The oil has escaped from the diseased synovial sac along the tendon sheath of the quadriceps, hamstrings, and gastrocnemius. This is the probable route of exit of infection from the joint into the surrounding tissues giving the peri-articular swelling and infiltration seen in many acute and subacute cases. (Case 4.)

ulation of the joint another film may be made to show the passage of oil into other portions of the synovial pouch and occasionally into bursæ Fig. 4).

It was found in one of our cases that the iodized oil had penetrated the synovial capsule and had passed into the tendon sheath of the quadriceps extensor (Fig. 8). We believe that the oil finds its way out through the synovial capsule because of synovial erosions. It has occurred to us that this is probably the route of extension of infection from the joint and that we may, in this way, explain the tendon sheath thickening and peri-articular induration which is seen in so many cases of acute or subacute arthritis.

For the injection of the hip joint a point is selected one inch below the tip of the greater trochanter and just anterior to it. The needle is introduced and follows along the neck of the femur until a definite obstruction is reached. This is the head of the femur. The needle is slightly withdrawn, pointed sharply anteriorly and directly into the joint cavity.

We have injected the knee joint in 7 cases and the hip joint in 3. Roentgenograms of some of these are shown.

In Case 5 a very faint shadow of the popliteal bursa is visualized. The oil had not penetrated the bursa sufficiently to outline it. This is undoubtedly due to the fact that synovial fluid so filled the bursa that the oil could not immediately pass into it. In those cases in which the bursa is at no time visualized, we have reason to believe that the bursal sac is either entirely obliterated or does not communicate with the free joint cavity.

Five days after injection a synovectomy was done in this case. The synovia was greatly thickened and studded with papillary formations and villous growths. Much of the iodized oil was free in the joint. We were much interested to know to what extent the oil had invaded this thickened synovial membrane as this might have a definite bearing on the therapeutic value of the injections in infected joints. The report by the pathologist, Dr. Hueper, reads as follows: "In sections with Sudan III stained fat droplets are seen adherent to the surface and in the intercellular spaces of

Fig. 9. Fig. 10. Fig. 11.

Fig. 9. Extensive synovial hypertrophy. Joint injected with 100 cubic centimeters iodized oil. Showing the enormous distention of the suprapatellar pouch divided into two separate compartments. (Case 5, M. S.)

Fig. 10. Lateral view showing distention of synovial pouch and a faint outline of a very large popliteal bursa. (Case 5.) At operation the bursa was found greatly enlarged with thickened walls and full of a straw colored fluid, the same as that which had been aspirated from the joint. Oil could not enter into the bursa because of presence of this fluid.

Fig. 11. (Case 6.) Synovial tuberculosis of the hip joint. Note partial filling of hip joint and the reflux of iodized oil into a portion of the tuberculous subtrochanteric bursa, the major part of which had been removed by operation.

the loose connective tissue and between the fibrils of the strong fibrous tissue. Fat droplets were also found in the lymphatics of the deeper layers."

Through a small opening in the popliteal space the popliteal bursa was exposed and completely removed. The walls were greatly thickened and infiltrated. The lumen was small and completely filled with the same type of fluid which had been aspirated from the joint cavity, but very few oil globules were seen. This bears out our contention that the presence of the fluid would not permit the oil to find its way into the bursa.

Clinically, beneficial effects have been derived from these injections of antiseptic oil into the joints in the majority of our cases. Freedom of action of the joint and relief of pain have been reported by the patients.

The results from this preliminary clinical investigation have been so gratifying that we feel fully justified in continuing these injections both for the purpose of diagnosis and therapy.

THE OPERATIVE TREATMENT OF EMBOLISM OF THE LUNGS[1]

PROF. DR. A. W. MEYER, BERLIN, GERMANY
From the City Hospital of Berlin-Charlottenburg—Krankenhaus Westend

IN Germany, the treatment of embolism of the lungs has interested physicians ever since the days of our surgical genius, Trendelenburg. But since Kirschner's first and only success (in 1924) the treatment has resulted only in failure. Quite recently, a number of patients have been saved, and it is of these that I wish to write.

Embolism of the lungs is a sort of Damoclean sword, which, unfortunately, threatens every patient who must lie in bed inactive for shorter or longer periods of time. It is not fully realized that such sudden emboli of the lungs occur not only after operation by a surgeon or a gynecologist, but that death from embolism of the lungs is just as frequent in the clinics for internal diseases, if one may be permitted to use the word frequent at all in speaking of embolism of the lungs. It was only since we began to study this strange and so often fatal sickness (thrombosis and embolism) that this fact became plain.

In the municipal hospital, Charlottenburg-Westend, we wished to perform on a dead body, an operation for embolism of the lungs, and while in one whole month, the surgical service, which has 500 beds, did not lose one patient from embolism, the medical service, which has the same number of beds, lost 7 patients. This should be of interest to all physicians, as well as operating surgeons.

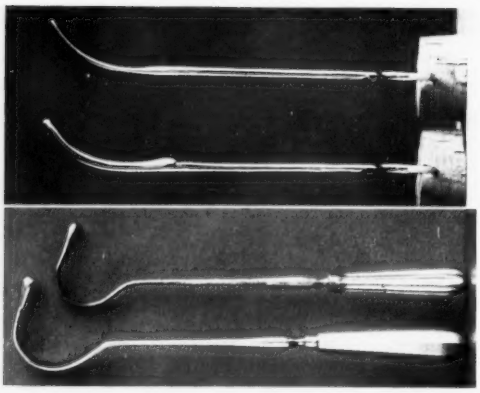
Since we have no sure means of avoiding a thrombosis and embolism, we are unfortunately certain to be confronted again and again with the severest cases of embolism, and faced with the decisive question: Shall we wait or not? Will the embolism be overcome? Is it one, anyhow? Shall we undertake the operation for embolism of the lungs? The severest symptoms of embolism can, of course, abate of themselves, and several times I have waited for hours by the bedside of a patient suffering from an embolus, everything prepared and ready, and fortunately did not need to operate. It is said that it is very difficult to know when to operate and when not. One who has closely studied patients with this sickness, and who has watched the effects of treatment, devoting himself wholeheartedly to the work, giving of his time and patience—such a man finds it easy to decide when to operate. The patient simply begins to die! And when the man of real experience sees that the patient is dying, he knows that it is right and proper to operate for embolism of the lungs. It is difficult to characterize the indications for an operation for embolism of the lungs any better; it is not a thing that can be described.

The decision to operate must be made as quick as lightning in the sudden cases, but more time may be used in the chronic, spasmodic cases of embolism. Once operation is decided upon I would advise the following procedure.

In 1908, Trendelenburg proposed that lung emboli be removed from the pulmonary artery. The steps of his procedure are: A T-shaped incision of the skin above the left rim of the sternum and the second rib; resection of the second rib, whereby the pleura is at once laid bare; section through the cartilage of third rib; and incision of the pericardium. After the pericardium is laid open a sound is passed around the great vessels (aorta and pulmonary artery) which are confluent and arise from the base of the heart. The Trendelenburg sound, which has a bayonet closure, must be connected with a rubber tube. The tube must be drawn through and around the vessels, which can now be drawn forward and strangled. According to the experiences of Trendelenburg, the period of strangulation may be 45 seconds but not more. The pulmonary artery is incised, the emboli are extracted from both large branches; a clip is applied on the side of the pulmonary artery, whereby the slit in the artery is squeezed together and it becomes possible to place the sutures above the clip. With this original Trendelenburg method of operation, Kirschner in 1924 saved the life of a patient, a woman of 38 years, and was thus the first to demonstrate that this bold and splendid procedure could be used successfully on human beings and might succeed in saving lives.

Up to this time, that is to say from 1908 to 1924, all attempts had been in vain. No patient had lived longer than 5½ days. When they did not die on the operating table, as was almost always the case, they died during the next few days as a result of subsequent loss of blood, infection of the pleura and the pericardium, or as the result of a pneumothorax. Since the Kirschner case, I know of numerous other attempts to operate, for example by Sauerbruch, but all were unsuccessful. I, myself, performed the operation on bodies on the dissecting table, and later tried to save a

[1] Lecture presented before the Mayo Clinic, Rochester, Minnesota, New York Post Graduate Medical School and Mount Sinai Hospital, New York.



Figs. 1 and 2. Original Trendelenburg clip and sound and author's modifications of each. The upper figures in each picture are the author's modifications.

patient who was dying of embolism of the lungs. This first, unsuccessful operation showed me that (1) the opening of the pleura implies the infliction of a stupendous shock upon the already injured heart; (2) the Trendelenburg sounds and clip must be improved, as will be shown; and (3) the strangulation period of 45 seconds caused by the introduction of the rubber tube around the great vessels puts too much strain upon the already laboring and dilated heart (a sound heart may be able to withstand this period) and that this period of strangulation evidently induces a paralysis of the respiratory centers which becomes irreparable.

I began dissection experiments in order to find out if the pulmonary artery could not be reached through an extrapleural route. I soon discovered that this could be accomplished in spite of the fact that Trendelenburg stated that he had discovered from his exhaustive studies, that it was impossible to avoid the laying open of the pleura. Kirschner also said that, in spite of the extreme danger of infection and the formation of empyema, one must of necessity open up the left pleural cavity, however unpleasant the one-sided open pneumothorax might be in the case of a patient fighting hard for his life.

I constructed a pulmonary artery clip with weaker and narrower branches so as to restrict the side stream of blood as little as possible (Fig. 1). I had the Trendelenburg sound made of a smaller size as the original model caused great inconvenience when it was inserted (Fig. 2). I also covered the pulmonary artery clip with gauze instead of rubber. With these simple improvements, I was able to save the next two victims of embolism upon whom I operated.

In the first patient (54 years of age), 6 days after a gynecological operation, there were signs one morning of a severe embolism of the lungs—sudden decline, cyanosis, pallor, labored breathing; pulse barely discernible. The Trendelenburg operation was contemplated but was not considered to be necessary as yet. At 2 o'clock the physicians on duty were summoned with the cry: "The sick woman is dying!" Her daughter, who was with her, rushed to meet us with the cry: "Help, help, my mother is dying!" No pulse-beat could be felt and the patient breathed with the utmost difficulty. She was chalk-white, and no longer reacted. Operation was started within 2 or 3 minutes. A T-shaped incision was made from the second to the third rib, extensive resection of these ribs was done, and the pericardium was cleared from the pleura and the mammary artery. The pleura was as thin as a spider's web, so that the lung could be seen moving beneath it. A slit was made in the pericardium and the Trendelenburg sound was introduced. The pericardial fat was scraped from the pulmonary artery which lay still, broad, and with-

Fig. 3, left. Specimen removed from first patient.
Fig. 4. Photograph of first patient showing scar from operation.

Fig. 5, left. Specimen removed from second case.
Fig. 6. Photograph of second patient.

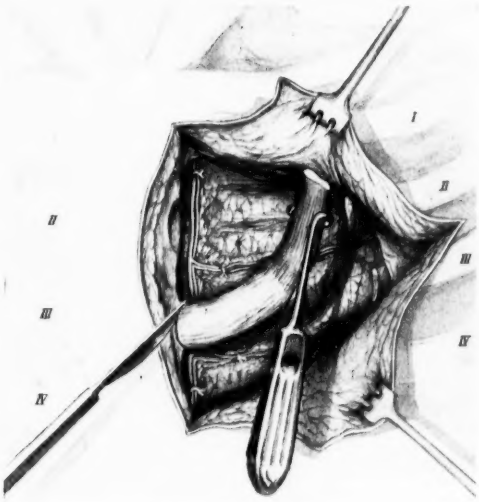


Fig. 7. Excision of rib.

Fig. 8. The forefinger of the right hand gently pushes under the insertion of the fourth rib.

out pulsation. The pulmonary artery was incised, and the sound was thrust rapidly three times into the right branch. No more emboli could be found on the third insertion. The heart then beat much more feebly, and the breathing became more labored. At this tragic moment the operator had a happy inspiration and squeezed the slit in the pulmonary artery between the thumb and forefinger. This made it possible for the blood to circulate more freely, because the rubber tube could now be released. At once there was improvement in the heart-beat, and in respiration. A few seconds later, as the heart action had greatly improved, slight tension was placed on the tube, and triple investigation of the left branch again resulted in the removal of large emboli. The pulmonary artery clip was fixed on the side. The heart began to flutter and respiration ceased. Gentle massage of the heart with the fingers

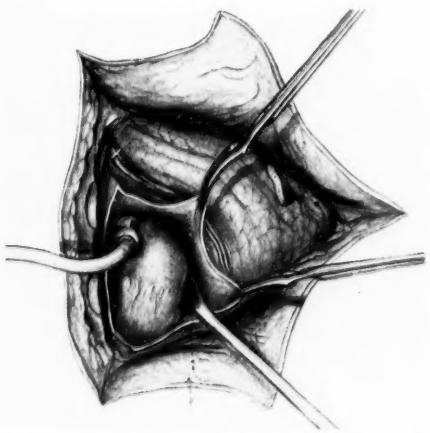


Fig. 9. Modified Trendelenburg sound introduced and rubber tube being passed around pulmonary artery.

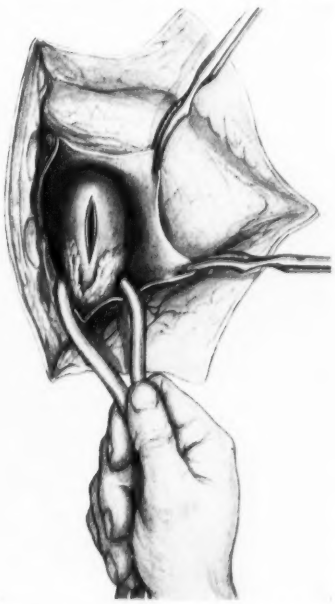


Fig. 10. Trendelenburg rubber tube in place

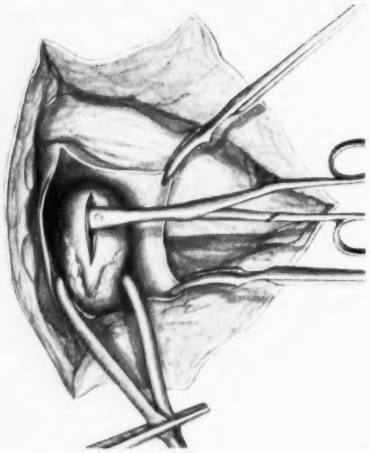


Fig. 11. Trendelenburg forceps are almost horizontal.

induced a few faint beats but respiration remained totally inhibited. The sick woman lay like a corpse, entirely without reaction. At the moment when the operator was about to order artificial respiration, my first assistant, Dr. Djialoszynski, suggested giving carbon dioxide, according to Henderson's method, and just as experiments had shown us, the patient drew a deep breath. A heart beat followed, then another, and the heart once more beat regularly. We could now sew up the vessel. The pulmonary artery clip was removed. Blood escaped at one point, so the clip was put back and another suture was made. This overcame all bleeding. The pericardium, muscle, and skin were sutured. When we started to suture the skin, the sick woman had revived sufficiently to cry out in pain, and consciousness returned. Her color was perfectly satisfactory, when she was taken back to bed. After a number of anxious days she made a complete recovery. Figure 3 is a picture of the specimen and Figure 4 of the patient.

The successful outcome in this case was followed a few weeks later, by another successful result after a modified Trendelenburg operation.

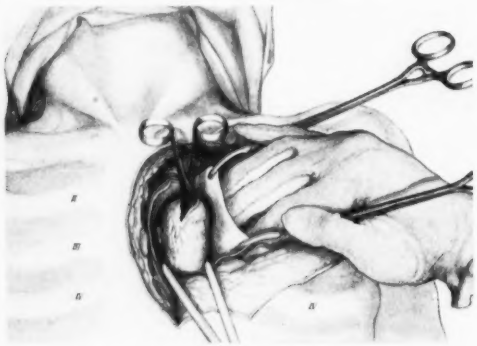


Fig. 13. Vertical exploration for emboli.

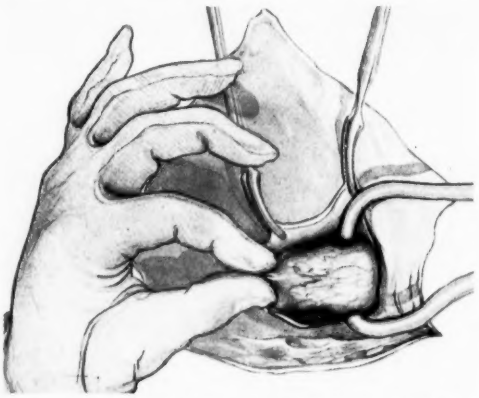


Fig. 12. Incision in artery grasped between thumb and forefinger to close slit.

One day when I was in the midst of an operation, a woman, 60 years of age, dying of an embolism, was wheeled into the operating room. She had been operated upon 24 days previously for gangrenous appendicitis, and embolism of the lungs had suddenly set in. In a few minutes I was able to do an embolectomy and even though it seemed that we might be too late, we saved the patient (Figs. 5 and 6). The woman lived nearly 4 weeks after this operation, making the best possible progress. She was about to leave her bed, I was unfortunately absent, when a new embolus caused her death. Postmortem dissection showed that the embolus had come from the other leg. The sutures on the pulmonary artery were perfectly healed. No thrombosis had arisen from them, so that one might say that the method was successful in this case also.

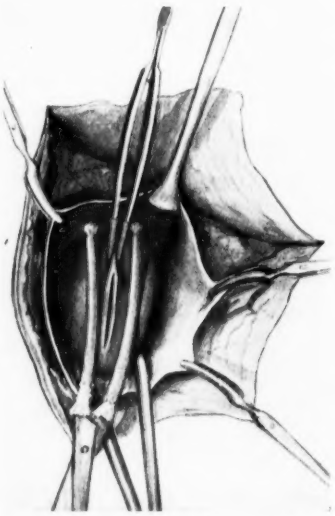


Fig. 14. Pincers covered with gauze on cranial end of slit.

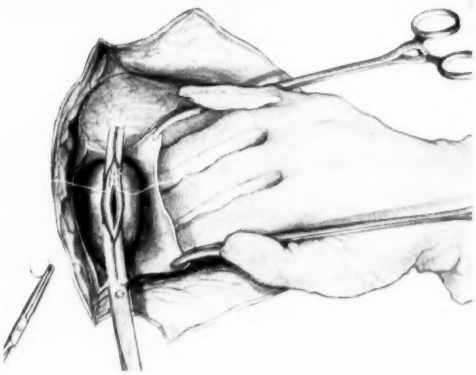


Fig. 15. Suture of incision commenced.

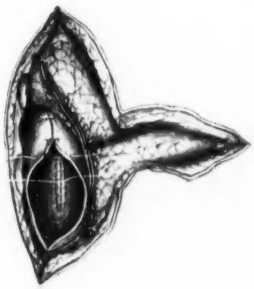


Fig. 16. Suture of pericardium.

TECHNIQUE

The steps of the procedure may be given as follows:

Since one is operating upon the dying, the operation is almost bloodless. The second and third rib must be laid bare with swift, large strokes of the scalpel. These ribs must be freed from periosteum and pleura quickly but very cautiously (Fig. 7). The operator now gently pushes away the mammary artery and the pleura. The forefinger of the right hand is inserted to enter by way of the lower medial angle, partly under the breast bone and the operator's hand gently feels its way under the insertion of the fourth rib (Fig. 8). The pericardium, shining white and partly covered with fat, is plainly in view. Incision with the knife is followed by a flood of pericardial fluid. The pericardial cavity is laid open by introducing cautiously and energetically both forefingers which are spread out. Thus the pleura is pushed still farther out of place, and the pericardial cavity can be opened to a surprising extent.

The next step is the insertion of my modified Trendelenburg sound and the placing of the rubber tube around the artery (Fig. 9). On the cadaver one may be in doubt as to which is aorta and which is pulmonary artery. In the living, or rather the dying body, there can be no doubt—the aorta lies hidden and the pulmonary artery, swollen and pulseless, is at once visible. The Trendelenburg rubber tube (Fig. 10) is useful, as we shall presently see, not so much in strangling the vessels as in bringing the large vessels out of the depths, which may be considerable in the case of fat patients, so that they can be better observed. The pulmonary artery is incised, the tube being held lower. A quantity of blackish blood rushes out. Emboli, which may come from the heart, are also flooded out. Greater tension is now applied to the rubber tube. Forceps are placed in the right pulmonary branch, and the Trendelenburg embolus forceps is now almost horizontal. If the right branch is found to be free of emboli, a triple investigation suffices, then the incision in the pulmonary artery is grasped with the thumb and forefinger of the left hand and is pressed together. The tube at once becomes absolutely relaxed. The blood is allowed to flow through the pulmonary artery for a few seconds, when, for the first time, the tube is fairly energetically tightened, and an almost vertical, triple penetration is made into the left branch (Fig. 13). The heart is then relieved by means of renewed digital compression of the slit in the pulmonary artery, with the tube quite relaxed. Now for the second time, the tube is forcefully tightened. The left hand grasps the cranial end of the slit vessel with a pincers covered with gauze, and holds it aloft (Fig. 14); the right hand attaches the arterial clip on the side; the tube is released. Any tightening of the tube naturally renders the work of the heart more laborious and thus injures it. The assistant must be cautioned regarding this, and must be ready instantly to obey the command: "relax," "more relaxed," "somewhat tighter," as the case may be.

I have given you an exact description of the manipulation of the rubber tube, because I believe that the respiratory center is very sensitive and should be shut off as little as possible from the arterial blood supply. It has recently been contended that strangulations can be endured as long as 60 seconds. I consider all lengthy strangulations to be dangerous for the nerve centers and those operators who trustfully make use of them will

Fig. 17. Instrument case. Instruments are arranged in the order of their use.

certainly be disappointed. The digital compression of the slit in the artery and at the same time the keeping of the tube as slack as possible—it is really only necessary to tighten it forcefully as the artery clip is attached—are certainly the least dangerous methods of procedure. In my third successful case, strangulation was reduced to a minimum and respiration though feeble did not cease for a moment.

The following case shows how sensitive the respiratory center may be: A woman who had a fracture of the leg was brought into the operating room looking like a corpse; she did not breathe and the heart had ceased to beat. Since I am an optimist, I attempted to extract the embolus precisely according to my modification, whereupon the heart, which, as stated, was quite still, began to beat slowly to the astonishment of us all, and then to beat so quickly and violently that the artery clip on the side had to be held to prevent its being hurled away from the heart. We thought that we might save the woman, but the respiratory centers reacted neither to carbon dioxide, Lobelin, or any other measures, and after artificial respiration had been practiced for an hour, the heart flagged and collapsed and nothing could be done.

This experience shows how sensitive the respiratory center is, and therefore, how important it is to have the strangulation of the vessels as short as possible. When respiration has become regular and the heart beat stronger and more rapid, then suture of the vessel can be undertaken deliberately (Fig. 15).

In such instance the heart pulsates sturdily; respiration is good; the face of the sick person, which was previously of a deathly white, becomes rosy; the muscle vessels begin to bleed; they are ligated, and the pericardium, muscle, and skin are sutured; and so the seemingly magic transformation of a patient from death to life in the course of a few minutes is accomplished.

Figs. 18 and 19. Photographs of clots removed and of patient showing scar.

The successful outcome in these 2 cases led us to give further attention to technique of this operation. We constructed an instrument case, which contained all the necessary instruments for the operation (Fig. 17). When this case is unrolled, everything is in readiness to proceed. The nurse in attendance hands out one instrument after another, just as they lie in the case.

The next patient with embolism of the lungs came to us a year later. This case was especially interesting, because it demonstrated the value of patience and perseverance. To sit many hours at a time by a patient's bedside is by no means pleasant, but it is sometimes well repaid by the results. It shows us that when an embolus is once on the move, one has a right to demand of an operating surgeon today that he should have a practiced hand and should be prepared to wait patiently and possibly to perform an operation after a delay of many hours. On four occasions we have waited hour by hour—once in the Charlottenburg Maternity Hospital. This patient developed a severe embolism about 8 days after childbirth. The obstetrician urgently desired an embolectomy, for according to their experience, which was undoubtedly extensive, they had never seen a patient recover after such a severe case of embolism in child-bed. In my opinion, however, the patient was not a dying woman, and this proved to be true for she made a good recovery without operation. If one does not keep watch by the bed of such a patient, however, it may happen that the fatal jerk upward may happen just when he is absent, or that the over-

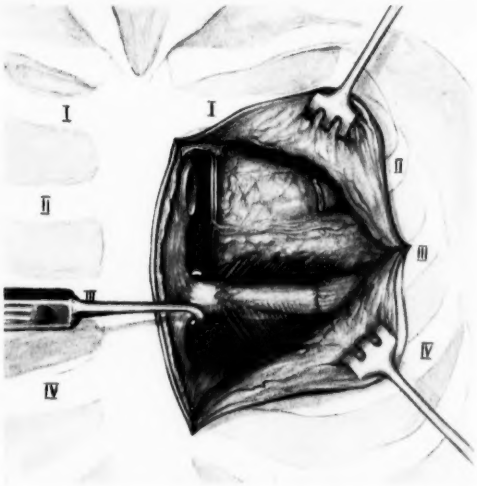


Fig. 20. The raspatory is introduced close to the sternum at the cartilaginous portion of the second and third ribs.

burdened heart may suddenly collapse while the surgeon is away.

The third case in which I did a modified embolus operation with success was that of a patient who was operated upon for a ruptured ovarian tumor. The wound healed splendidly. Ten days later at 6 o'clock in the morning, signs of a very severe embolism manifested themselves. The sick woman continued to recover under continual watching until at about 6 o'clock in the afternoon a renewed, severe clotting ensued. Still I was not at all inclined to do an embolectomy. However, I continued to keep watch by the sick woman's bed until 12:15 that night, when the heart-beats became fainter and fainter, the breathing more and more dyspnœic, and consciousness evidently began to vanish. The patient's last words were: "I think I'm going to die." Her pulse could scarcely be felt, respiration most arduous and superficial, and she looked like a dying woman. We did the typical operation as already described. The wounds healed without any further incident (Figs. 18 and 19).

As I have already said, up to now, only Kirschner has been successful with the original Trendelenburg method (resection of the second rib, transpleural procedure), and I believe that he was successful only because his patient was young and her sound young heart was able to withstand the enormous burden laid upon it by the pneumothorax.

It gives me great joy to say that two surgeons in Sweden, who had heard my lecture and had seen my demonstration at the Surgeons' Congress in 1926 and who had previously not been successful when they used the original Trendelenburg method, have now had splendid results when they followed my method precisely. These two

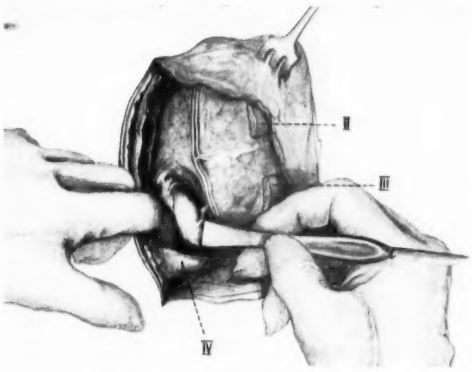


Fig. 21. Pushing away fat with handle of scalpel and pleura with forefinger of left hand.

Swedish surgeons have each had two successful cases. These with my 3 cases, make 7 cases reported in which operation has been successful.

Unfortunately, I experienced a great disappointment a few months ago. A very large man of 50 years of age contracted an embolism of the lungs after a simple appendectomy. The extrapleural clearing and the opening of the pericardium were accomplished according to our usual technique, but on account of the unusual depth in which the organs lay, as the patient was very fat, I had the misfortune to injure the heart in introducing the Trendelenburg sound. This had previously happened to others, among whom might be mentioned Trendelenburg himself, and Sauerbruch. This accident caused me to make many further experiments on the dissecting table, and I can now present the steps of an operation which is so abbreviated and simplified that it can be carried out without difficulty by any surgeon or gynecologist, even by those who are not familiar with the technique of resection of the ribs. Previously, subperiosteal resection of the ribs with a pleura often as thin as a spider's web, was a task, especially in a moribund patient, extremely trying. The operation must be carried out with meticulous care and yet with great rapidity. With my technique, however, one can resect the ribs in a few seconds and the steps of the operation are simple and the pleura is not opened or injured. This technique has not previously been described. The sternum must be exposed more than usual. It is not resected extra-pleurally as usual at the bony portion of the ribs. The raspatory, however, is introduced quite close to the sternum at the cartilaginous portion of the second and third rib (Fig. 20). This takes only a few seconds. Fat lies

Fig. 22. Incision into pericardium.

Fig. 23. The incision is slowly but forcefully widened with the two forefingers.

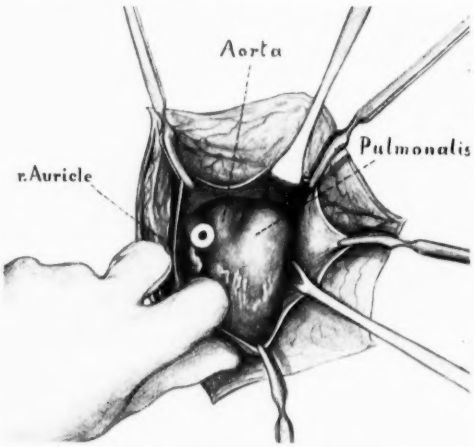


Fig. 24. Right auricle exposed.

beneath, loose, and can readily be pushed aside. Here the Doyen raspatory is brought into play. It is inserted medial to the internal mammary artery and when pushed further in a lateral direction we resect the ribs extrapleurally as far as necessary and without danger to the pleura, because in this layer, the pleura is surprisingly easy to remove. Even in the case of the most aged subjects, the operation is done with undreamed-of rapidity and certainty. With a large bone forceps three nips, about ½ a centimeter broad, are made in the sternum at the level of the second and third rib. The fat and pleura under the sternum have previously been set aside with the raspatory. The reversed handle of the scalpel is used to push away the fat of the epicardium and the pleura on the right side, and the left forefinger is used to push away the pleura on the left (Fig. 21). Immediately the pericardium comes into view, it is brought up between two rigidly bent pincers and an incision is made and then widened digitally, as I have already described (Fig. 22). The two forefingers slowly but very forcefully widen the pericardial slit, the left proceeding cranially as far as the first rib, the right as far as the fourth rib (Fig. 23). The space now is very large. The right auricle is plainly visible and it is at once apparent how easily and quickly the small model of the Trendelenburg sound can be introduced (Fig. 24). Injury is no longer possible. We have not as yet had a chance to operate on a dying patient with this method, but I am convinced that this further improvement means considerable simplification of the operation—something which doubtless will make the operation popular. From inquiries and replies from many surgeons, it would seem that the bad results from operation are due to the fact, that even by extrapleural procedure, overhasty operators tore the pleura. With my modification this can not happen.

I am convinced that, since no means has yet been discovered of obviating postoperative thrombosis and embolism, the Trendelenburg operation will be more and more used and that it will be practiced by other operators just as is any other operation. The operation is not so difficult as it would perhaps appear. I therefore hope that my suggestions will induce others to try my technique and that they will be successful in relieving patients suffering with this fatal malady.

PROLAPSE OF THE INTESTINE THROUGH A PREFORMED OPENING IN THE GREAT OMENTUM

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THE finding at operation of one or more apertures in an otherwise unremarkable omentum majus is not a rarity. Such unsuspected slits are generally found at the termination of an abdominal operation. They may, of course, be due to injury incidental to the operation at hand; but again, in some instances, they are noted before operative interference and when found, are repaired, and prove of no further significance.

That a slit or slits of this sort may form the starting point for some intra-abdominal catastrophe, such as the ensnaring of a segment of intestine, appears reasonable. The occurrence of such an event, however, from a review of the literature, appears extremely rare, and it is difficult to say anything concerning its actual incidence. Makins, in a study of 400 cases of intestinal obstruction, reported one instance (0.25 per cent) wherein an opening in the omentum was the cause of an obstruction. No description is given of this case.

We are concerned, in this discussion, only with openings in the omentum that are apparently preformed and not obviously the result of inflammatory adhesions. This, therefore, excludes those instances in which adhesions between the margin or other portions of the omentum to each other as well as to contiguous structures result in constricting bands or what at times appear to be abnormal apertures.

Concerning the etiology of omental openings, there is probably more speculation than actual information. From the appearance of one of these slits it is probably impossible to determine whether the abnormal opening is of recent or remote formation. It would appear rather arbitrary to consider such openings congenital unless they are formed in the early period of life; in fact, Prutz believes that the most common cause of omental openings is the gradual atrophy of the connective tissue.

From a review of the available literature the following personal observation forms, we believe, the sixteenth case record of its kind.

E. A. L., a white woman, aged 24 years, was first seen October 29, 1927. The family history was altogether irrelevant. The past history was entirely negative except for the following notations. For several years the patient had had more or less "abdominal gas" with a sense of epigastric fullness immediately after meals. Belching gave relief, and there was no history of pain or constipation. One year ago she had "bladder trouble," which lasted 3 days. This was not treated by a physician and was not associated with abdominal symptoms.

The menstrual history was irrelevant. The patient had been married 8 months.

The present illness began 9 days previously at which time the patient began to have chilly sensations. Two days later she experienced acute epigastric discomfort with nausea and vomiting. This discomfort became generalized to the entire abdomen but was most noticeable in the right iliac quadrant. The discomfort was constant, dull, and aching, with exacerbations which were not colicky-like and did not radiate. There was no further nausea or vomiting, but the chills persisted as did the abdominal discomfort and for 2 or 3 days there was urinary frequency and burning.

Physical examination. The temperature was 99 degrees F.; pulse, 64; respiration, 14. The entire examination was irrelevant except for the abdominal findings. The abdomen was symmetrical and moved freely with respiration. At first there was such marked cutaneous hyperæsthesia in the right iliac quadrant that it was almost impossible to touch the patient who was very apprehensive. However, when the examination was finally satisfactorily performed, there was elicited generalized abdominal discomfort on palpation and percussion but no rigidity or muscle spasm. There was also some discomfort to pressure in either costovertebral angle. No viscera were felt, the flanks were tympanitic, and the area of hepatic flatness was normal. The pelvic examination was irrelevant.

A provisional diagnosis of pyelitis was made with appendicitis as a possibility.

In the hospital the patient's temperature was never above 98.6 degrees F. The white blood count on two occasions totaled 9,800 and 7,000, with 70 per cent and 60 per cent polymorphonuclear neutrophilic leucocytes respectively. The blood Wassermann reaction was negative. A urine specimen obtained by urethral catheterization contained numerous leucocytes and gram positive cocci. A cystoscopic examination with bilateral ureteral catheterization was irrelevant except for a very moderate hyperæmia of the bladder mucosa, while microscopic preparations and cultures of the urine from either kidney showed just a few leucocytes and gram positive cocci in stained smears of the dried sediment. The cultures showed a staphylococcus.

The abdominal symptoms persisted and in spite of the evidence of a pyelitis it was obvious that the pyelitis was not producing either fever or leucocytosis, and therefore was not the cause of the abdominal symptoms.

Operation was performed November 3, 1927. An appendix, normal both macroscopically and histologically was removed. A complete exploration was also irrelevant until at its termination the reflected omentum was turned down for interposition between the parietal and visceral peritoneum. It was then seen that a loop of intestine lay upon the anterior aspect of the omentum. Closer inspection revealed two openings in the omentum in about the midline, the smaller one superior to the larger as shown in Figure 1.

The smaller opening was oval, measured probably 1.5 to 2 centimeters in its greatest dimension, and was situated about 2 to 3 centimeters inferior to the transverse colon.

CASES FROM THE LITERATURE

Authors' Name Case	Date	Age of patient	Sex	Portion of intestine involved	Symptoms	Outcome	Remarks
No author's Name 1	1840	49	F	Small	Typical of obstruction	Death on second day. No operation	Patient had cancer of uterus. *Autopsy:* some small intestine had protruded itself through the omentum and become strangulated—"almost gangrenous."
Curran, J. W. 2	1869	12	F	Small?	Typical of obstruction	Death on 16th day. No operation	Child previously well. Frightened by dog; ran and while running felt violent pain in "belly." *Autopsy:* "Good sized rent" in omentum behind umbilicus with "good sized knuckle" of gangrenous intestine protruding through. Peritonitis.
Piere, A. 3	1872	..	M	?	Typical of obstruction	Operation. Recovery	Loop of intestine had passed through an aperture, 3.5 cm. in extent, of "accidental, traumatic, recent origin" situated close to the free border of the great omentum. No gangrene. Nothing in description to show its traumatic origin.
Cayley and Lawson 4	1877	47	M	Small (ileum) "40 inches" long	Typical of intestinal obstruction.	Operation. Death. Cause of obstruction not found at operation	*Autopsy:* 40 inches of small intestine "bluish black" had passed through an opening, 3 inches in circumference, in "right lower angle of omentum." Omentum free. No adhesions.
Uhde 5	1882	..	..	Small	.....	.....	Author merely mentions a case of von Krombholz of intestinal obstruction due to loop of intestine passing through opening at margin of great omentum.
Berléme-Nix 6	1886	27	M	Apparently small gut. Length not given	Typical of intestinal obstruction. Fecal vomitus. No abdominal signs	Operation. Recovery	Patient had had previous similar attacks. Omentum full of holes; resected. Intestine incarcerated and easily released. No previous operation.
Cattell, H. W. 7	1896	56	M	Small. 10–12 feet	Typical of obstruction	Death. Findings at autopsy	Intestinal strangulation. Bloody fluid in peritoneal cavity. Omental opening not localized in description.
Guinard, A. 8	1898	24	M	Small?	Typical of obstruction	Operation. Recovery	Tuberculous peritonitis. Omentum appeared as a "sieve" 10–15 "hernias" in these holes one of which was incarcerated. No previous operation.
Gorski, K. 9	1898	82	F	Transverse and descending colon	Typical of intestinal obstruction	Operation; aspiration of large intestine; recovery	Opening, 4 cm. in diameter, at edge of omentum. Edge of ring about 2 cm. in thickness and of almost cartilaginous consistency. Considers his case one of old peritonitis and the defect of inflammatory origin rather than a simple tear in the omentum.
Barling, A. S. 10	1898	21	M	Small?	Strangulated inguinal hernia	Operation. Recovery	Omentum and intestine in hernial sac. Strangulation due to intestine passing through a hole in the omentum. Necessary to cut omentum to free intestine.
Lisanti, A. 11	1899	..	M	Small	Typical of obstruction. Tumor felt to left of umbilicus. *Diagnosis:* Intussusception	Operation; resection of 25 cm. small intestine; Murphy button anastomosis; uncomplicated recovery	Patient had syphilis 5 years previously. Loop of small bowel compressed by opening in great omentum. Bowel gangrenous. Considers omental defect of luetic origin because of small nodules and areas of ulceration with grayish-yellow bases about defect.
Goodman, Chas. 12	1906	63	M	Small; 8 inches long	Typical of obstruction	Operation. Recovery	Intestine strangulated due to volvulus. Incarcerated in omental opening, one inch from free border. Reduction by cutting omentum. No previous operation.
Taylor, Wm. 13	1907	..	..	Small?	.....	.....	Author reports 35 cases of intestinal obstruction. One of these a case of intestine passing through an opening in the great omentum which "was evidently of recent formation." No further description given.
Datta, J. K. 14	1918	43	M	Small; 6 feet long	Acute upper abdominal pain 5 minutes post cibum. Slight distention with tenderness and general rigidity. No fever. *Diagnosis:* Perforated gastrointestinal ulcer	Operation. Recovery	Large amount of blood stained fluid in peritoneal cavity. Intestine prolapsed through aperture in great omentum at the free border. Gut slightly dilated, dark purplish, not gangrenous. No adhesions.
Makins, G. H. 15	1919	..	..	....	.....	.....	No description. Merely a reference to an instance of intestinal obstruction due to an opening in the omentum.
Martzloff, K. H. 16	1929	24	F	Ileum	Indefinite. No pre-operative diagnosis	Exploratory laparotomy. Recovery	See text.

The large defect, also oval, measured probably 5 to 6 centimeters and was situated about 2 centimeters inferior to the smaller one. A loop of small intestine (ileum I believe) protruded through the larger defect and lay upon the omentum to the right of the midline. The loop measured about 15 to 20 centimeters in its total length, was slightly distended, and of a purplish-pink hue. Its mesenteric vessels were questionably dilated though the subserosal capillaries were visibly enlarged. Reduction was easily accomplished and the intestine showed plainly the two points where the omental constriction had been effected.

The margin of the two omental apertures showed no evidence of hæmorrhage or fibrin deposition. They were smooth and covered with a pale gray, glistening, and translucent serosa—evidently preformed defects of undetermined age.

The defects were repaired. The patient had an uneventful recovery and is well at the present time. There have been, however, occasional recurrences of the bladder symptoms.

This case differs from the others encountered in a review of the literature in that here the outstanding symptoms of intestinal obstruction were absent. It also illustrates the ease with which a symptom producing anatomical abnormality may be overlooked in an otherwise "obscure case" unless the exploration is performed systematically and, where possible, under direct vision.

In the accompanying table are arranged in chronological order the reported cases of intestinal prolapse through a preformed omental opening. From this table have been omitted, as previously noted, instances in which omental adhesions were the source of intestinal ensnarement. Particular mention may be made of the case reported by Brown which Prutz apparently accepts as coming within the group of cases we are considering. From Brown's description the omental slit was evidently due to an adhesion on the posterior aspect of the omentum. The intestine in his case had passed through an aperture that was posterior to the omentum. In other words there was no transomental defect and we have therefore not included this case in our tabulation. The cases of Atkins, Fowler, and McWhorter also do not come within the scope of this paper. There may be some doubt as to the propriety of including the case reported by Gorski. This was one of an opening 4 centimeters in diameter at the *edge* of the omentum through which almost the entire transverse and descending colon had passed. The edge of the ring was about 2 centimeters in diameter, and Gorski expressed the belief that the defect was inflammatory in nature rather than a simple tear in the great omentum. This is a most unusual case and probably belongs to the group we are studying.

It also appears that in his monograph, Prutz accepts the case reported by Möller. In this instance the omental opening was caused by the fusion of two pieces of omentum. The findings were made at autopsy which occurred 23 days after a previous abdominal operation and we are excluding this case from our tabulation.

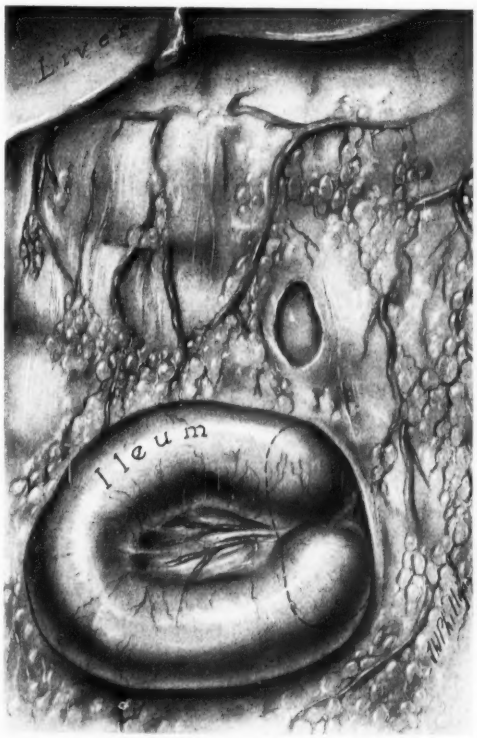

Fig. 1. Drawing showing location of openings in the omentum.

From a comparison of the case reported by Cayley and Lawson from the Middlesex Hospital in 1877 with that of Coupland in 1879, we believe the two reports concern the same patient. In each description the omental defect is localized in the same situation in identical words, the amount of small intestine that passed through the omental aperture was "40 inches" in each description and in both observations the site of the distal strangulation was "4 inches from the ileo-cæcal valve." We, therefore, have considered this a duplicated case report and have given priority to Cayley and Lawson.

A case reported by Kirchner also deserves mention. Here the jejunum had passed through the omentum but in his careful description Kirchner explains that the constricting bands were pro-

duced by adhesions of the margin of the omentum to the pelvic organs. This case has, therefore, been excluded from our tabulation.

The tabulation is self-explanatory. The number of reported cases is so small that one doubts the permissibility of any conclusions utilizing percentage figures. The following summary may serve to crystallize the few generalizations that may be drawn from this study.

SUMMARY AND CONCLUSION

1. Prolapse of the intestine through a preformed omental defect not due to adhesions is an exceedingly rare occurrence. In so far as we have been able to determine from the literature this case report represents the sixteenth instance.

2. It may occur at almost any age and there is not sufficient information available to indicate its predilection for any particular age period.

3. No definite predisposing factor has been determined.

4. The small intestine is the portion generally involved.

5. The symptoms of this condition are, as one would naturally anticipate, typical of intestinal obstruction. In the case here reported the foregoing generalization is not applicable.

6. The outcome following timely operative intervention has been uniformly good.

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MEDIAN COLOSTOMY

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SINCE colostomy was first employed for the purpose of providing an artificial anus, the left inguinal region has been almost invariably selected for its location. The sigmoid colon has been employed for this purpose more often than any other portion of the large bowel.

An abdominal opening over the point where the largest portion of this bowel is most often found, was the most natural one to be made. Inasmuch as an artificial anus functions best when the fæces which pass through it are normal in consistency, shape, and quantity, as a general rule, the lower in the colon it can be located, the better it is.

The rectus muscle being thick and strong not only has made a good support for the bowel, but has exercised, at least to a slight degree, some sphincteric function.

It is gratifying to find so many patients who have been subjected to a colostomy in possession of so marked a degree of fæcal continence as to be able to attend to their regular duties. In a large number of cases, however, the fæcal discharge from a left inguinal colostomy has irritated the surrounding skin so that much discomfort has been suffered by the patient.

In people of slight build or those who have been emaciated as a result of their disease, the anterior superior spine of the ilium, being quite prominent, becomes eroded, ulcerated, and is made very sensitive. On account of this discomfort, dressings, bands, belts, trusses, or colostomy apparatus could not be worn without increasing the patient's distress. This in many instances has created a problem the solution of which has been attended with considerable difficulty.

Patients forced to go through life with a colostomy and who must wear some sort of a bag or other retaining device are usually quite sensitive about their condition. Usually bulging or protrusion on the left side caused by colostomy pads, cups, or bags, causes such a distortion of the patient's contour as to be decidedly noticeable and embarrassing. This is particularly so in people who have become emaciated through illness. In women especially, the difficulty in arranging the clothing so as to disguise their infirmity has presented serious difficulties.

In performing a left inguinal colostomy, the surgeon is limited in most cases to the employment of the sigmoid colon for the colostomy. The gastrocolic omentum does not allow of sufficient laxity of the transverse colon, without undue strain on the stomach, to be brought down and successfully used in this region.

Before performing a colostomy, obviously a complete exploration of the abdominal cavity is necessary for both positive and negative diagnostic reasons. An incision either at or near the median line must be employed to make a thorough, complete, and successful intra-abdominal surgical examination.

If one elects to perform a left inguinal colostomy after such an examination, a second incision and opening into the cavity must perforce be performed. If, as is often the case, a colostomy is performed preliminary to a subsequent abdominal or abdominoperineal extirpation of a malignant growth of the rectum or sigmoid colon, it is quite important that the colostomy be placed not only as high up in the bowel as possible, but also that it be located as high up on the abdominal surface as possible.

By employing the descending or the transverse colon in some cases one is able to resect a larger portion of the colon above the growth and thus secure a greater margin of safety. By placing the colostomy well above the site of the wound which would be necessary for a subsequent resection, a clean area is provided for the abdominal operation.

For some years we have been locating our colostomies in the median line, just above the umbilicus, and our patients have been able to wear colostomy bags with much greater ease than when the colostomy was located in the inguinal region.

One unsatisfactory feature in this location has been the soiling of the umbilicus by fæcal discharge and the difficulty in cleansing this area. For this reason during the past 3 years we have been excising the umbilicus and using this site for the location of the colostomies.

The technique as first advocated by Angelo Soresi has been employed with slight modification in certain cases. By employing a natural opening into the abdominal cavity which the umbilicus provides, we take advantage of the semicircular arrangement of the rectus fibers at this point as well as of the increased strength of the fascial tissue which lessens the tendency to possible later herniation.

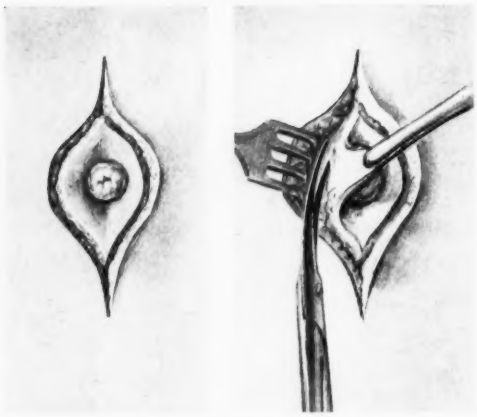


Fig. 1, left. Line of incision.
Fig. 2. Freeing the umbilicus.

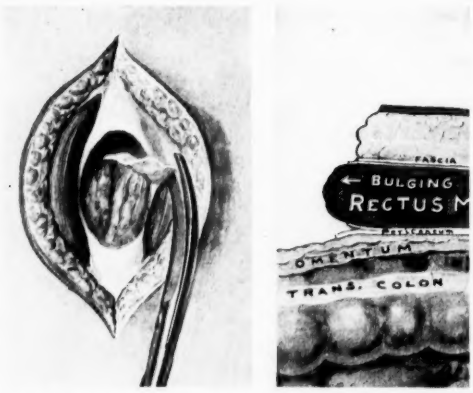


Fig. 3, left. Area punctured for insertion of rubber tubing.
Fig. 4. Sectional view.

The technique of operation as personally employed is as follows:

If local anæsthesia is used, a one-half per cent solution of novocain in Ringer's solution is chosen. A subcutaneous injection is made from two punctures located at both poles of the proposed incision. This is very rarely longer than 3 inches and is made in the median line curving outward on both sides around the umbilicus, above and below, and then again joining above. The fascia is punctured and the muscle infiltrated (Fig. 1) on either side down to the peritoneum. Care must be taken not to puncture the peritoneum for fear of injuring the bowel. The umbilicus is grasped with an Allis forceps and freed right down to the peritoneum with scissors and by blunt dissection. Through a peritoneal incision just below the umbilicus, an examination (Fig. 2) is made in order to be sure that omental adhesions or protruding intestines are not present. With the removal of the umbilicus, a sufficient opening into the peritoneal cavity is thus presented so that a complete ocular examination can be made.

By extending the incision downward and continuing the injection of the anæsthetic solution, and using great gentleness, we can locate the neoplasm. Mesenteric traction must be avoided to prevent unnecessary pain. The liver, gall bladder, and spleen can be palpated through a similar extension of the upper half of the incision. With rubber-tipped forceps a portion of the descending or transverse colon is brought out of the wound. A non-vascular area of mesentery (Fig. 3) is punctured with hæmostatic forceps and a piece of ¼-inch thick-walled rubber tubing brought through this mesenteric opening to act as a support for the colon for the first few days. If the transverse colon is used, sufficient omentum is tied off in sections, so as to give a clean loop of bowel for the colostomy (Fig. 4). The fascia and peritoneum are trimmed back on either side so as to allow the rectus muscle fibers to bulge into the wound. The peritoneum is closed above and below the bowel by plain catgut sutures and the muscle and fascia approximated by number two interrupted chromic catgut sutures. No attempt is made for the purpose of bringing the peritoneum up to the skin as recommended by some authors.

Much better muscular control is obtained by the adhesion of muscular fibers directly to the intestine. The skin is closed by clips and stearate of zinc powder or sterile vaseline applied freely over the protruding bowel.

Much more satisfactory anæsthesia for this operation is that obtained by the subdural injection of novocain. Spinal anæsthesia produces such a wonderful degree of intestinal quiesence that having once employed it in any type of abdominal surgery, the surgeon is prone to use it always. The utter flaccidity of the abdominal wall as well as its contents renders intra-abdominal examination of all of the viscera a matter of greatest ease. Any operation, and especially on the colon, is facilitated by a complete absence of intestinal movements which is so characteristic in etherized patients. The intestines are contracted and lie on the posterior wall of the abdominal cavity, as they do in the cadaver.

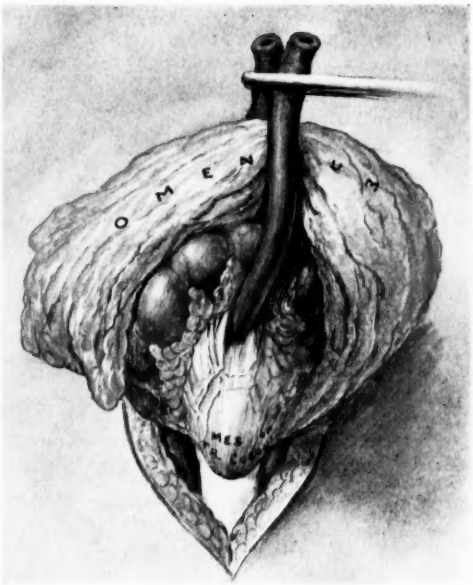


Fig. 5.

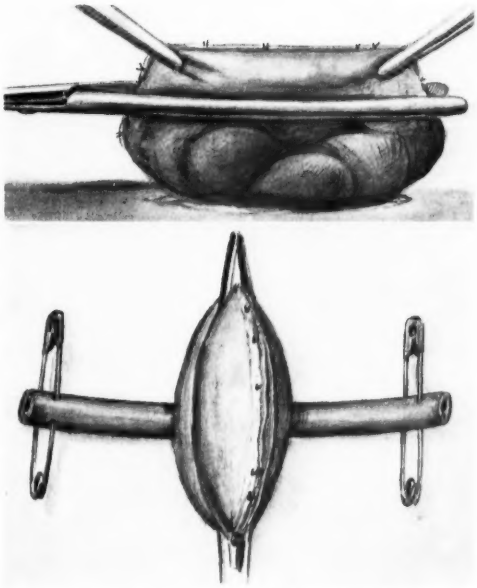
Fig. 6.

The operative technique under spinal anæsthesia is the same as when local anæsthesia is performed. There being no protrusion of the intestines, it is not necessary to use gauze or other substances as packs or coffer-dams, therefore no unwelcome intestinal adhesions are produced.

For several years, a very simple and satisfactory method of opening the colostomy loop has been personally employed. Noting the repugnance of the patients to the odor of their own burning flesh, the cautery has never been employed for the purpose of opening the colostomy. Incision is attended by unnecessary hæmorrhage and is also disturbing psychologically, even though it is painlessly performed.

Usually the opening of a colostomy means a second trip of the patient to the surgery or operating-room, which is also often unnecessarily disturbing. For this and other reasons, opening the colostomy by means of pressure necrosis has been personally employed with very satisfactory results (Fig. 7).

After the operation described has been completed and before the dressing is applied, the exposed loop of colon is seized with two pairs of hæmostatic forceps grasping the longitudinal muscular band about 3 inches apart. Traction is used to raise this portion up while a long curved



Figs. 7 and 8. Colostomy is opened by means of pressure necrosis. This method gives very satisfactory results.

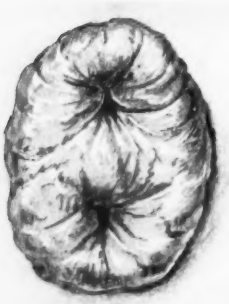


Fig. 9. Colostomy completed.

hysterectomy clamp is placed along the bowel just below the ends of the traction forceps. The clamping of this forceps puts pressure on a spindle-shaped area which becomes the opening of the colostomy, when the clamp is removed after 48 hours.

In this way a painless, bloodless colostomy opening is produced without the patient being aware of it. The opening is satisfactory and is produced while the patient is in his own bed and it does not require any special preparation, instruments, or "fuss and feathers."

The skin surface surrounding the colostomy is kept protected with a large quantity of stearate of zinc powder, not only during the healing process, but afterward. The patient is encouraged to sit up in bed just as soon as the abdominal incision is sufficiently healed to allow the removal of clips or stitches. He is then urged to attempt to have a movement from the colostomy at regular stated periods, usually morning and evening.

If no movement is secured, the colon is flushed through the colostomy, with a few ounces of saline or soda bicarbonate solution. By holding a crescent basin under the colostomy with one hand while with the other, he irrigates with a bulb syringe, the patient is able to take care of himself nicely without assistance.

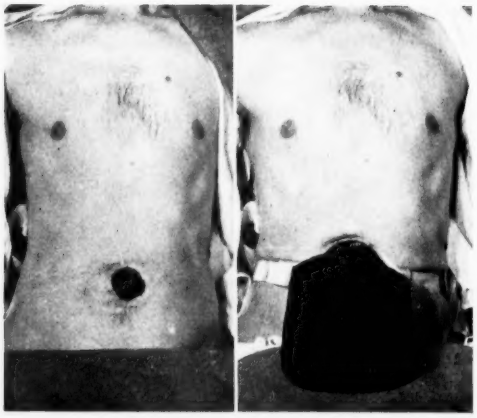


Fig. 10. Photograph of patient showing colostomy and belt with removal bag.

A colostomy belt with a removable bag is used, which is very light in weight and which is inexpensive. This has been found much more satisfactory than the more expensive and complicated types of apparatus. The patient is encouraged to walk as soon as he is able and to get out of doors and resume his normal routine of life.

I fully believe that a median colostomy is more strongly supported by surrounding tissues, than is one located in the inguinal region. On account of this fact, the convalesence is more rapid and the patient is able to resume his activities in a much shorter period of time, which is a good thing from a psychological standpoint as well as a physiological point of view.

IMPLANTATION MALIGNANCY OF THE ABDOMINAL WALL

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THE fear of local recurrence of malignant tumors prompts the surgeon to excise the initial lesion as widely as he dares. Invasion of the abdominal wall by extension from intra-abdominal malignancy is not so unusual and is an occasional sequel to advanced tumors of the large bowel. Implantation of neoplastic tissue into the abdominal wall during excision of an intra-abdominal neoplasm, without its recurrence within the abdomen is, however, a very unusual occurrence. A careful review of the literature has borne out this impression. The rarity of this occurrence justifies the report of the following case.

Mrs. M. W., 69 years of age, white female, housewife, was admitted to the University Hospital in May, 1923, complaining of a tumor of the anterior abdominal wall.

The patient was a healthy individual throughout her youth, suffering only a few of the common illnesses of childhood. Her menses appeared at the age of 13 years, and were regular and normal in every respect. She was married at 23. She was never pregnant. Her menses continued normal until she reached her middle thirties when they appeared every 2 to 3 weeks. Pain and profuse vaginal bleeding was noticed then on the first day of each menstrual period. This caused her to seek medical advice and treatment.

In 1907 at the age of 48 years she had erysipelas of the face for several weeks. In the winter of 1907–1908 she visited her physician because of menorrhagia and was told that this was due to change of life. In April, 1908, a curettage was performed and a small fibromyoma of the uterus was removed. Following this she developed increasing leucorrhœa. In June, 1908, the appendix, right ovary and right tube were removed. In November, 1908, the uterus, left ovary, and left tube along with a cyst and two tumors of the uterus were removed. These latter were found to be large fibroids. The abdominal wound became infected and healing was somewhat slow. Dr. Ray Knight, who assisted at these operations and who saw the patient from time to time, has kindly given us the information relative to her operations. Dr. F. R. Woodard, who performed the original operations at the old Asbury Hospital, Minneapolis, is deceased and the hospital records of that time are not available.

The patient continued apparently healthy until 1921 when she first noticed a lump about the size of a walnut in her left lower abdominal quadrant. At first it grew slowly and then more rapidly until May, 1923, by which time it had developed to the size of a small football. Shortly, thereafter, she was admitted to the University Hospital. At this time she was suffering from sharp, burning pains at the site of the tumor mass, occurring four to five times a day but not radiating in any direction. Her best weight had been 180 pounds, but she had lost 17 pounds during the 6 months previous to her admission here. She also complained of some weakness. The family history revealed that only one relative, a great aunt, had died of some form of cancer.

The physical examination showed, on palpation and inspection, a hard, diffuse mass about the size and shape of a small football, extending in a slightly oblique direction from a point 5 centimeters below and 9 centimeters to the left of the umbilicus to a point on the pubic crest 3 centimeters to the right of the midline. This mass was slightly tender on pressure and appeared embedded in the musculature of the abdominal wall, because it could be moved about freely with the abdominal wall and also because the skin could be easily lifted away from the mass. Three centimeters to the right of the midline, and extending upward from the pubis to a point half way between the latter and the umbilicus, was a scar from the operations of 1908. Beneath this scar the abdominal wall appeared rather thin and to it the right edge of the tumor was fixed. It was felt that this tumor was probably a desmoid of the rectus sheath, and excision was recommended. No adenopathy could be demonstrated.

The tumor, including a large part of the left rectus muscle and its anterior sheath, was removed on July 27, 1923, by Dr. A. L. Cameron. On microscopic examination the tumor was found to be an adenocarcinoma, its structure closely resembling alveolar glands lined by columnar epithelial cells. Convalescence was satisfactory and she was discharged apparently well. During 1924 she received superficial X-ray treatments for her abdominal wall condition as prophylaxis against recurrence.

However, the patient was readmitted to the University Hospital on June 26, 1925, because of recurrence of the tumor and abdominal pain. There was found over the site of the previous operation a large postoperative incisional hernia. In the abdominal wall, about 2 centimeters to the left and slightly below the umbilicus, a round, hard, tender mass, about the size of the patient's fist, was palpable. Over the pubic region was found a hard, nodular, irregular mass, roughly 8 by 5 by 5 centimeters in size, adherent to the pubis and tender on palpation, first noticed in September, 1924. The adnexa were negative. The pelvic floor was normal. The rectal examination was negative. The patient complained of numbness and pain in the left lower extremity, which radiated from the sole of the foot to the thigh. She had lost 20 pounds in weight since the fall of 1924 and had become progressively weaker. On July 12, 1925, 900 milligram hours of radium in needles were inserted into the suprapubic mass.

On October 29, 1925, the patient was readmitted for the fourth time. The suprapubic mass was smaller, she felt better, and she said that she did her own housework. The mass near the umbilicus, however, was somewhat larger. The following day she was given 35 milligrams of radium for 10 hours into the suprapubic mass, a total of 350 milligram hours, and advised to return later for superficial X-ray treatments of the umbilical mass. She was very co-operative and appeared regularly at the out-patient clinic at intervals of about 4 weeks. Each time she received approximately 40 per cent of a skin erythema dose to the mass near the umbilicus. Treatment was continued until the summer of 1926, when she said she felt good. The abdominal wall tumor had become smaller. The new Cancer Institute, at the University of Minnesota, having now been fully established as a unit of the University Hospitals, deep X-ray therapy was begun. Between August,

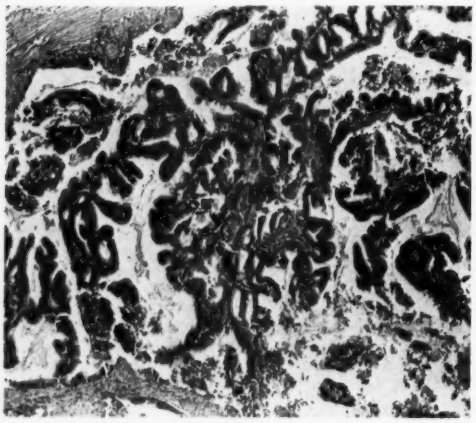

Fig. 1. Photomicrograph of ovarian cystadenocarcinoma removed at time of necropsy from the abdominal wall of our patient.

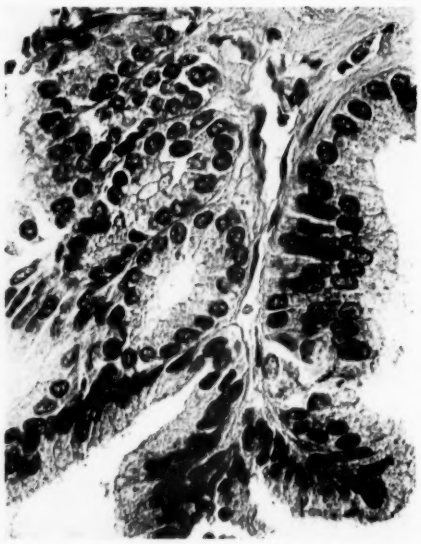

Fig. 2. Same as Figure 1, under higher magnification ×450.

1926, and March, 1927, she received three series of deep X-ray therapy.

On June 7, 1927, she was readmitted for the fifth time. There were many small nodules, which could be felt on palpation, slipping around beneath the skin of the lower abdomen. She felt quite well except for a dull pain in the back which was now more or less constant. Her appetite was good, she had no pain in the abdominal masses and felt fairly strong. On June 13, 1927, X-ray examination revealed metastases involving the fourth and fifth lumbar vertebræ, which accounted for the pain in the back.

Further deep X-ray therapy was recommended. This was instituted and by August, 1927, the abdominal wall tumor near the umbilicus could scarcely be palpated and she again felt much better. Altogether, she received between June 23, 1927, and December, 1927, three series of deep X-ray treatments, receiving each time a 110 per cent skin erythema dose to the central part of the pelvis and spine. In January, 1928, the scar tissue surmounting the postoperative incisional hernia and tumor area began to break down. By April, 1928, following nearly a week of obstinate constipation, an intestinal fistula developed, opening onto the abdominal surface.

The patient was admitted to the Minneapolis General Hospital in August, 1928, with a history of mental confusion for 2 days and constipation for 4 days preceding admission. She complained of shooting pains about the pelvis radiating to the umbilicus. The clinical impression was generalized carcinomatosis. A history note of October, 1928, mentioned the persistence of a large, hard abdominal mass on the left side. There was considerable emaciation at that time. Death occurred on November 26, 1928.

The postmortem examination revealed a pear-shaped abdominal defect, 10 centimeters by 8 centimeters immediately above the symphysis pubis. In the abdominal defect were two fistulous openings, one connecting with the ileum at a point 50 centimeters from the ileocæcal junction, the other with the midportion of the transverse colon. The intestines in the upper part of the abdomen were bound down by adhesions. The peritoneal surfaces were free of metastases. The pleural and pericardial cavities were free of metastases. No lymph node involvement could be demonstrated. However metastases were found in the fourth and fifth lumbar vertebræ. The bones of the pelvis appeared normal. The organs of the head and neck were not examined.

Below and to the left of the umbilicus was a fleshy tumorous mass 5 centimeters by 2 centimeters in the left rectus muscle region. Microscopically, the tumor was found to be composed of many atypically and poorly formed alveolar structures lined by tall columnar cells and supported by a thin stroma. In many of these alveoli the lining cells had proliferated to the extent of forming fine dendritic processes projecting into the lumen, occasionally forming the point of separation of one partly formed alveolus from another. The picture as a whole was that of a papillary cystadenocarcinoma, very likely of ovarian origin. The following anatomical diagnoses were made: (1) emaciation and cachexia, (2) hypostatic bronchopneumonia, (3) implantation carcinoma in the abdominal wall with metastases to the spine, (4) intestinal fistulæ.

It is believed that the origin of the neoplasm in the scar of this case can be attributed to implantation of a portion of the supposedly benign ovarian cyst into the abdominal wall incision at the time of operation in 1908.

In contrast to implantation in the abdominal wall extraperitoneally, a much commoner condition is the well known intraperitoneal dissemination from a benign ovarian cyst with implantation upon the peritoneal surface. This process was probably first recorded in Baker Brown's celebrated case which was described by Beigel in 1869.

Wagner (1864) probably described the first case of benign ovarian tumor reproducing its structure

in the abdominal wall proper. A former prostitute, who over a period of 13 years was tapped 42 times because of ascites, had developed three subcutaneous cystic tumors below and to the left of the left breast, in the right axillary fossa, and in the left flank region. These tumors were secondary to the very large intra-abdominal benign papillary serous cyst arising from the left ovary. The histological structure of all was identical, as proved at necropsy.

Baumgarten (1884) mentioned the extirpation of a benign papillary serous ovarian cyst. At necropsy sometime later, secondary tumors, all histologically benign and identical with the primary ovarian cyst, were found to have occurred not only on the inside of the peritoneum, but also in the abdominal wall extraperitoneally.

Olshausen (1899) described the instance of a woman aged 46 years who had had a left oophorectomy performed in 1878. In 1895 she was operated on by him for an abdominal wall tumor of the right side just above the navel. Histologically, the latter was a benign papillary ovarian cystadenoma. The author believed this to have arisen undoubtedly by inoculation of the abdominal wall during the operation done 17 years previously.

In the same paper this author mentioned another case of a woman of 53 years, who in 1889 was operated upon for the removal of an intraligamentary cystic ovarian tumor, the size of a man's head. On section the tumor was found to have a papillary structure. In 1895 she was operated upon for recurrence in the right ovary and also for a fist sized abdominal wall tumor which proved to be carcinoma. The author believed that these latter were entirely secondary to the first tumor of 6 years previously.

In 1902 Olshausen again reported another similar case of a woman, who in 1881, at the age of 29 years, had been operated upon for bilateral tumors of the ovary (bilateral ovarectomy). These were of the papillary cystic type. For the past half year she had noticed a tumor on the right side of the abdominal wall, which progressively increased in size. It was removed and diagnosed grossly as unquestionable carcinoma, a histological diagnosis not having been ready at the time of the report. This the author believed arose by inoculation at the time of the previous operation because the abdominal wall tumor was found to arise from the scar of the previous laparotomy.

This last case demonstrates three interesting facts found together which had occurred separately in previous cases, namely: a long interval of time elapsing between the removal of the primary benign ovarian tumor and the appearance of the secondary in the abdominal wall; origin of secondary in the scar of the previous laparotomy wound; finally, malignancy of the secondary tumor.

Schnuetgen (quoted by Tauber) up to 1918 found only 8 published cases of benign ovarian tumor "metastasis" to the abdominal wall. To these he added a case.

Bland-Sutton (1922) removed the uterus and bilateral, apparently benign, papillary ovarian cysts of a woman. Six years later a rapidly growing tumor of the sternum on the right side at the level of the second intercostal space was noticed. It was removed but promptly recurred and eroded away the whole manubrium. This secondary tumor was microscopically exactly like the primary cyst. The patient died. At necropsy, strangely, no recurrence appeared in the abdomen.

Tauber (1927) reported a woman whose abdomen began to swell and fill up with fluid in 1919. She was finally explored; fluid was removed. It was not known whether a tumor had been found. Her ascitic condition recurred and she was reoperated upon in 1921 when bilateral papillary ovarian cysts were removed together with three smaller ones found on the peritoneum of the anterior abdominal wall below the umbilicus. No trace of malignancy could be found. In 1926, the patient returned with another recurring cystic mass around the umbilicus and entirely within the abdominal wall. This was removed. Histologically, the tumor was found to be a malignant ovarian cyst.

Lang mentioned a pseudomucinous cyst which was removed from the left ovary of a 30 year old woman. Two months later a tumor developed in the scar of the previous laparotomy wound, which proved to be an adenocarcinoma.

The cases described were all of the pseudomucinous or papillary cystadenomatous type. However, a similar course of events has been reported occurring in another type of tumor. Jaquet, in 1899, described an ovarian dermoid which was removed from a woman in 1874. Seventeen years later, 1891, an abdominal wall tumor 15 centimeters in diameter appeared, which proved to be cancerous.

These cases, 10 in number including our own, were all that could be found in the literature in which primary ovarian cysts, supposed to be benign at the time of their removal, were later followed by a benign or malignant reproduction of their general structural type in the abdominal wall (in the thoracic wall in Bland-Sutton's case). Of these 10 new-growths, 6 were observed to be

malignant and 4 benign. The secondary growths were first noticed 21 years after discovery of the primary growth in 1 case, 17 years in 2 cases, 13 years in our case, at least 10 years in 1, 7 years in 1, 6 years in 1, 2 months in 1, and 2 were of undetermined duration. Of the 6 malignant tumors, one appeared 21 years after discovery of the primary lesion, one 17 years, our case 13 years, one 7 years, one 6 years, and one 2 months.

In all the cases the primary ovarian cysts were considered benign when extirpated. Cases have also been reported in which the primary was obviously malignant when extirpated and simultaneously presented secondary malignant metastases to the abdominal wall, specifically to the umbilicus. Cullen (1916) collected 9 such cases.

Mayfield (1926) summarized a study of 100 cases of papillary cystadenoma of the ovary and found that these cysts vary both pathologically and clinically in their degree of malignancy. He found that some small cysts were very malignant, and some large ones benign. Benign and malignant areas coexisted. Such variations in degree of malignancy may account for the great variations in time interval between removal of primary and appearance of secondary abdominal wall growths found in the 10 cases in this series. Papillary ovarian cysts, especially when bilateral, always arouse a suspicion of malignancy and though no malignant area may have been found microscopically, the feeling frequently obtains that it may have been missed. Bell (1924) says: "Recurrence and metastases of ovarian papillary cystadenomata is frequent, even from tumors which appear anatomically benign. All papillary tumors are potentially malignant." For these reasons it is believed that the most likely explanation in the case reported here is that the ovarian cyst removed 20 years prior to the patient's death was malignant from the beginning, even though that fact was not observed or demonstrated at the time.

In locating the primary source of implantation tumors of the abdominal wall, lesions of the gastro-intestinal tract and uterus must be considered as well as of the ovaries. The mere fact that ovarian cysts had previously been removed must not be misleading. Polano (1905) collected from the literature reports of 7 cases of malignant abdominal wall tumors arising in laparotomy scars after previous removal of apparently benign ovarian tumors. He added one similar case. Necropsy in the latter revealed the presence of carcinoma of the stomach which he believed was very likely the primary growth. Of the 7 cases collected from the literature, on only one, that of Pfannenstiel, was necropsy performed. No intra-abdominal cancer was found here. Nevertheless, Polano considered the abdominal wall lesion in this case primary and not subsequent to the ovarian cyst. Because of the findings in Polano's case and because no necropsy was done on the other 6 cases, the suspicion always remained that the abdominal wall carcinomata in these 6 might easily have been secondarily implanted not from ovarian cysts but from carcinomata of some other abdominal organ which was missed at the time of the first operation or developed subsequently.

Brewer (1921) cites the case of a woman, who in 1911 had her uterus, tubes, and left ovary removed because of fibroids of the uterus. In 1920 she developed a tumor of the abdominal wall. Removal proved it to be a pure fibromyoma. The author explained its origin to implantation at the time of the previous operation because microscopically it resembled in all details the original uterine fibromyoma.

Adenomyomata, described as endometriomata, appear to have been rather frequently observed in the abdominal wall and umbilicus in recent years. Cullen (1920) described a case of an adenomyoma of the rectus muscle occurring in a woman who 8½ years previously had been operated upon through an abdominal wall incision to repair a ruptured uterus.

Also, Mahle and MacCarty (1920) mention 2 cases occurring in old laparotomy scars and also two cases arising, without apparent cause, in the umbilicus.

Lochrane (1923) mentions a case of adenomyoma implanted in the abdominal wall which appeared 4 years after ventrosuspension of the uterus. The tumor was always painful around the menstrual periods. This tumor contained typical uterine glands and smooth muscle. Previous to this (1916) Cullen had already collected 13 adenomyomata of the umbilicus, 4 of which were somewhat doubtful. Ewing (1919) also described cases of adenomyoma of the groin.

Lemon and Mahle (1925) reported 9 ectopic adenomyomata invading the abdominal wall after operation.

Nicholson (1926) reported a case of endometriosis occurring in an old laparotomy scar following a salpingectomy and mentioned 15 such cases reported in the literature following ventrosuspension of the uterus. Not in one case was an anatomical continuity established between the uterus and tumor thus showing that the latter must have arisen by implantation.

Lefievre and Montpellier (1927) described a case and mentioned more than 30 cases now on

record of an endometriosis of the umbilicus. Pratt (1927) discussed 42 cases now on record of implanted endometriosis occurring in old laparotomy scars following previous operations, the most frequent type of which was ventrosuspension of the uterus. He added 4 cases.

Sampson (1928) found only 1 case of endometriosis of the abdominal wall following ventrofixation of the uterus with tubal sterilization.

Jacobson (1926) working on rabbits showed that peritoneal implantation of endometrial tissue at a distance from the uterus was most successful during oestrus. From this it might be contended *a priori* that operations on the uterus in the human during or around menstruation are particularly subject to endometrial implantation.

Sampson (1925) offered circumstantial evidence in favor of malignant changes in endometrial tissue in the ovary as the source of certain ovarian cancers.

The literature examined revealed one or two cases of direct extension of a primary uterine adenocarcinoma into the anterior abdominal wall, but no implantations following operative removal of uterine carcinoma. Thus, it is seen that fibromyomata, more commonly adenomyomata (endometrioma) arising as primary uterine tumors may be implanted into the abdominal wall at the time of their removal. Such, however, was not the situation in our case.

For purposes of orientation I have included below a brief discussion of abdominal wall tumors as a whole. These divide themselves into two divisions: the abdominal wall in general and the umbilicus in particular.

Malignant tumors of the abdominal wall constitute only a very small proportion of malignant neoplasms. Gurlts (quoted in Zweifel-Payr) found among 16,637 cases of malignant tumors in general only 27 malignant tumors of the abdominal wall: 14 sarcomata and 13 carcinomata.

At the University Hospital of the University of Minnesota of 28,013 patients admitted between the years 1910 and 1923, only 9 cases of abdominal wall tumors were found. Of these, 7 were primary: fibroma 1, angioma 1, lipoma 2, hæmatoma 1, leiomyoma 1, sarcoma 1; two were secondary—both carcinomata by direct extension and not by implantation.

SARCOMA OF ABDOMINAL WALL

Of malignant tumors in the abdominal wall in general, both sarcoma and carcinoma are found. Sarcomata arise from the skin as well as from the fascia and muscle sheaths of the abdominal wall; they may originate malignantly *per se*, or benignly from a fibroma or nævus, particularly a pigmented nævus, which subsequently undergoes malignant degeneration. The different histological types found are fibrosarcoma, fibromyxoma, angiosarcoma, and, according to another classification, spindle, round, and giant cell sarcoma. The spindle cell sarcoma appears to be the most frequent of this group. Von Klot (1921) collected some 408 cases of abdominal wall connective tissue tumors of which he found 268 to be fibromata.

BENIGN TUMORS OF ABDOMINAL WALL

Of the truly benign types found here are fibroma, lipoma, angioma, myxoma, fibromyoma, adenomyoma, endometrioma. To these must be added rarer forms like atheroma, teratoma, and dermoid and echinococcus cysts. In this benign group, as von Klot's figures have already shown, the fibroma is not only the most frequent, but also the most important. This holds for abdominal wall tumors in general. Mueller (1838) first applied the name, desmoid tumor, to this group. According to Balfour (1916) they occur in women in the ratio of 7 to 1. The average age is 34 years. They are usually found in the anterior or lateral abdominal wall and in 43 per cent of the cases are associated with the rectus muscle, or its sheath, usually the posterior sheath. They are frequent after repeated pregnancies. They are usually of small size but may equal the size of an adult's head, are usually smooth unless very large, and are ovoid, the long axis in the direction of the muscle fibers. The cut surface shows a wavy white glistening surface of fibers intimately interwoven. Microscopically, they vary from a solid fibrous connective tissue overgrowth to a very cellular actively growing fibrosarcoma. The blood supply is poor as in other fibromata so that necrosis may occur. The etiology is obscure, or may be due to traumatic overstretching and rupture of the rectus muscle sheaths during pregnancy. Prognosis is good. Balfour reported 7 cases of which 2 occurred in previous operative scars.

With respect to the origin of desmoid tumors, Danforth mentioned work done by Loeb in which the latter incised the uteri of pregnant guinea pigs. Shortly, thereafter, he got nodular growths in the uterine scar. These could be elicited only in uteri which were pregnant. Such tumors can be considered analogous to desmoids caused by traumatic stretching of the abdominal wall in pregnancy.

CARCINOMA OF ABDOMINAL WALL

Carcinoma of the abdominal wall is either primary or secondary. Outside of the umbilical

region, primary carcinoma of the abdominal wall is rare. Secondary carcinoma of the abdominal wall is more common and a primary lesion should always be sought, especially in the gastrointestinal tract, liver, and female genitalia. The secondary lesions develop by direct extension, by metastasis, or by implantation at the time of previous operation. Such implantations have occurred not only in the scars of previous laparotomies, but according to Williams (1893), also at the site of puncture wounds after frequent tapping of ascites from an ovarian cyst.

DIFFERENTIAL DIAGNOSIS

Besides true neoplasms there must also be considered the inflammatory tumors of foreign body reactions, hæmatomata, rupture of the rectus muscle resulting in bulging of the ruptured ends and the infectious granulomata of tuberculosis, syphilis, and actinomycosis.

Abdominal wall tumors must be differentiated from intraperitoneal and retroperitoneal tumors. This may be done by having the patient lie on his back and having him raise his trunk to the sitting position without aid, thus contracting the abdominal wall muscles. If the tumor disappears completely from sight and becomes impalpable, it lies behind the abdominal wall. If it disappears only to sight but remains palpable and at the same time immovable, it is in the abdominal wall. If it is not at all influenced but remains visible and palpable, it lies either intracutaneously or subcutaneously. Abdominal wall tumors projecting into the abdominal cavity and tumors of intra-abdominal organs projecting outward become easily confused with one another.

Regarding the differentiation of intraperitoneal tumors from retroperitoneal tumors, Bevan (1924) emphasized the usefulness of inflating the large bowel with air and then percussing the abdomen over the site of the tumor area. If tympany is found, the colon overlies the tumor and the latter is retroperitoneal; if dullness is found, the tumor overlies the colon and the former is intraperitoneal. Use of the X-ray is always indicated.

TUMORS OF UMBILICUS PROPER

Regarding malignant tumors of the umbilicus proper, the sarcoma is considered the most frequent among connective tissue tumors. On the whole it is rare. More frequent is carcinoma of the umbilicus, which is both primary and secondary. Primary carcinoma is of the squamous and columnar type. The squamous type can arise from remnants of the epithelium of the urachus, or ductus omphalomesentericus.

Secondary carcinoma of the umbilicus may arise from any of the abdominal organs. Head (1926) reported an interesting case of primary carcinoma of the cæcum with metastases to the umbilicus.

Umbilical cancer, if primary, is usually of the squamous type; if secondary, it is of the columnar cell type and in that case an indication of an internal primary lesion. The histological structure may frequently indicate its exact location.

Of benign tumors there must be mentioned fibroma, fibrolipoma, angioma, myxoma, adenoma, infectious granuloma and inflammation of the abdominal organs.

SUMMARY

During the excision of an intraperitoneal malignant tumor, implantation into the abdominal wall may occur. Such an implantation may survive and at a more or less remote date begin to grow and present itself as a neoplasm of the abdominal wall. Such an occurrence is reported in this paper. Fifteen years after the patient's first operation, a tumor was removed from the abdominal wall which proved to be an adenocarcinoma on microscopic section. Following X-ray and radium treatment, the patient survived for more than 5 years. At necropsy metastases were seen in the spine (fourth and fifth lumbar vertebræ), but no source for the abdominal wall tumor masses was found. Only a few such instances are mentioned in the literature. After a careful search only 9 besides the 1 reported above were found. Most of the tumors originate from ovarian cysts. They are likely to be mistaken for dermoid tumors, as was the case in the instance reported here, unless the possibility of implantation is borne in mind. The subject of tumors of the abdominal wall in general is briefly discussed.

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EDITORIALS

SURGERY, GYNECOLOGY AND OBSTETRICS

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MAY, 1930

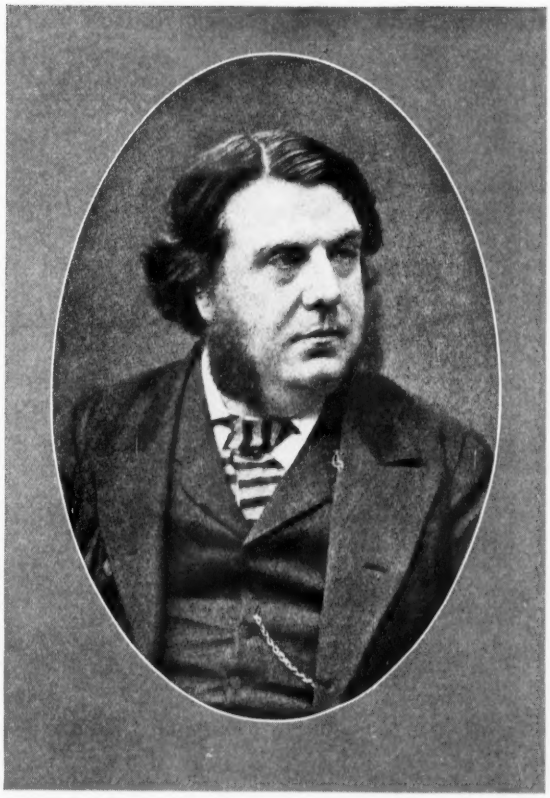
LAWSON TAIT

THE untimely death of Lawson Tait, of Birmingham, England, 30 years ago, cut short one of the most notable medical careers of the last century. That he was a genius no one can doubt who will make himself familiar with his contributions to gynecological and abdominal surgery. Though by no means the first to undertake serious operations within the abdominal cavity, he certainly did more to render abdominal section a safe and practical procedure than any other man and is fairly entitled to the honor claimed for him by Dr. William J. Mayo of being "the father of modern abdominal surgery."

Tait began his career as a laparotomist at a time when the operation of ovariotomy had been practically abandoned in England. Of the last 30 ovariotomies performed in Edinburgh, where Tait had received his medical training, not a single one had survived. The operation was actually forbidden in some of the leading London hospitals. Syme, the leading Scotch surgeon, one of Tait's teachers, to the end of his life refused to perform the operation. Tait nevertheless had the courage to undertake it soon after he began practice in a provincial town at the age of 23, and before he was 26 he had done the operation 5 times. At his death in 1899, at the age of 54 years, he had performed several thousand abdominal sections and with a degree of success unrivaled by any other surgeon.

I happened to arrive in Birmingham the morning of Mr. Tait's death from uræmic coma. I had had the privilege of spending a few months with him as a pupil assistant just 10 years before. On alighting from the train I directed the cabman to drive me to The Crescent, Tait's home and private hospital. Instead of doing so the cabman handed me the morning paper, which was in mourning and bore in large black letters across the page the announcement of Mr. Tait's death. Within a few hours the whole city was in mourning, for, next to Mr. Chamberlain, Mr. Tait was unquestionably its most distinguished citizen. His fame had brought to him suffering men and women from the ends of the earth. He had many patients from the United States and Canada and from South Africa and Australia. One patient, an American, the wife of a missionary doctor, suffering greatly with an enormous ovarian tumor, came from the remote interior of Burmah, having been carried several hundred miles on the shoulders of men to reach the nearest railroad station.

My first meeting with Mr. Tait was in his little office at The Crescent. He sat alone behind a small flat topped desk with a flexible speaking tube close at hand through which he dictated to his secretary in another room. As he sat in his chair he gave one the impression of being a man of gigantic proportions. His



LAWSON TAIT (1845–1899)

shoulders were very broad, his chest thick, and his large head—he wore a number 8 hat—was covered with a thick mass of dark hair which was inclined to curl. His neck was short. His strong facial features and his abundance of wavy hair gave him an almost leonine aspect. When he stood, however, the impression of greatness diminished somewhat as he was scarcely of medium height.

In manner Mr. Tait was kindly and courteous but rather short and abrupt. He had the air of a man preoccupied with intense thought. His speech was rapid and incisive, his sentences terse and pointed. He had an unusually large vocabulary and his choice of words was always the best possible. His ordinary speaking voice was pleasant, almost musical. When aroused and vexed, which often happened, he would roar like a mad bull. Tait was one of the most tender hearted men I ever met. He was gentle and delicate in his manner of dealing with patients and scrupulously careful to observe all the proprieties.

Tait had many crotchets and allowed prejudices to warp his judgment and blind his mental vision. He had a particular dislike for Emmett, one of the finest and sweetest of men. I could never discover any reason for this except that he disagreed with him respecting the nature of the pelvic inflammations to which Emmett had applied the term cellulitis. Tait believed the chief seat of these troubles to be the fallopian tubes, which subsequently turned out to be the truth. He carried his opposition to Emmett so far as to denounce everything he taught as error. In one case which I had previously studied at the dispensary and in which he was preparing to repair, after his rapid fashion, a torn perineum, there was also a badly torn uterine cervix. I asked, "But, Mr. Tait, are you not going to repair the cervix before closing the perineum?" "Oh, no," he said, "I never pay any attention to Emmett's little crack."

During the several months I was with him he never once repaired a torn cervix although cases of this sort came daily under observation. I doubt if he had any other reason, than his prejudice against Dr. Emmett, for thus ignoring lesions of the cervix.

Having some years before when in Vienna

(1883) become acquainted with Billroth's pylorectomy and Woelfler's gastro-enterostomy, I one day inquired of Tait why he did not perform these operations. He at once replied, "Pylorectomy is useless because it is never done except for cancer and the cancer always returns. I never do useless operations." The operation of gastro-enterostomy he condemned in equally strong terms, declaring that it always resulted in "continuous fæcal regurgitation." His attitude toward these operations illustrates one of the weak points in his character. When a prejudice was once established in his mind it was impossible to uproot it and it so blinded him that he was apparently incapable of treating the subject with intellectual fairness.

In a controversy Tait was a dangerous opponent. He was remarkably skillful in repartee and so dexterous a controversialist that he rarely failed to carry off the honors in discussions at medical meetings even when he was in the wrong. Tait enjoyed nothing better than lampooning an adversary, especially one whom he considered worthy of his mettle. On one occasion his opponent was a well known surgeon who, as his colleagues well knew, had for years been combating the inroads of Father Time by the adroit use of hair dye. In discussing Mr. Tait's paper the gentleman suggested that too much weight should not be given to his views because of the fact that he was known to be a man of very strong prejudices; whereupon Mr. Tait instantly retorted that he had only one prejudice in the world and that was against a man who dyed his hair. This savage sally quite annihilated his opponent.

Mr. Tait's animosity against some of his rivals was so great that it was hardly prudent to mention their names. On the one occasion of which I spoke to him of Spencer Wells he launched upon such a vehement outpouring of barbed criticisms and acrid animadversions I never ventured to mention his name again.

During operations Mr. Tait rarely spoke except to utter now and then a monosyllable or two by way of direction to a nurse or the anæsthetist. At other times, however, when riding with him in his carriage, as I had often an opportunity to do, or when riding on the cars, Mr. Tait was a genial and interesting conversationalist and had apparently an inexhaustible fund of information on any subject that might be broached. Although he did not finish his university course before beginning his study of medicine, his literary work during the early years of his residence in Birmingham as editorial writer for the *Morning Post* had led him into nearly every field of human interest. He had also been a student of biology under Darwin, whom he almost deified.

Mr. Tait frequently attended the theater, which he greatly enjoyed, although he often fell asleep and sometimes snored so loudly as to create considerable disturbance. When not occupied he was in fact liable to fall asleep at any time. In riding up to London I have known him to sleep for almost the entire distance sitting bolt upright in a corner of the compartment and snoring loudly. On one such occasion when the customary fog happened to lift for a few moments, allowing the sun to illuminate his face, I managed to get a good kodak picture of him. Later he allowed me to take another picture as he was in the midst of a surgical operation, his face wearing the intense and rather savage look which it usually had while he was operating. He was very much amused when I presented him with the two pictures mounted on a card labeled "Wide Awake" and "Fast Asleep." This was his first introduction to the Eastman Kodak, then just out, and he became the possessor of one as soon as possible.

Tait was not spectacular in his methods of operating, but in his work he was remarkably quick, neat, accurate, and efficient. His hands were large, his fingers short and thick, but remarkably deft. His precise, dextrous, and rapid movements in the performance of an operation was a fascinating spectacle—never a false movement, though he did some extraordinary things. For instance, if in making an incision a spurting artery made a pause necessary for the application of a ligature, he would often catch the handle of his knife between his teeth instead of handing it to an assistant or laying it down. He did everything himself. He rarely allowed the assistant to do anything more than to hold an artery forceps or to support a large tumor while he applied ligatures to the pedicle.

To the writer's knowledge, Tait has seldom been excelled in rapidity and dexterity. Dr. "Jimmy" Wood, who was the star operator in Bellevue Hospital when I was a student there in the seventies, used to cut off legs in 30 seconds, and Liston sometimes amputated thighs in 20 seconds. Martin, the famous Berlin gynecologist, did a double salpingectomy in 8 minutes. I saw Tait do the same operation in 7½ minutes. I often noted the time occupied in perineal operations and seldom found it more than 3 minutes, although McKay, who followed me in Tait's service, in his excellent biography makes his time for this operation 5 minutes. On one occasion I held my watch and saw Tait begin and complete an operation for partial laceration of the perineum in just 1½ minutes.

His ordinary method of operating on patients at the Spark Hill Hospital was this: With his coat off, sleeves rolled up, and wearing a big apron, he stepped to the side of the bed, seized the anæsthetized patient, and placed her crosswise on the bed with her hips at the edge, a nurse holding each limb. With a pair of tissue forceps in one hand and scissors in the other, he dropped upon his knees and with a few quick snips dissected the vaginal flap, made a deep cut on either side, seized a long-handled Peaslee needle, and pulled through three or four silkworm-gut sutures so placed as to secure good coaptation of the raw surfaces. The whole operation was over in little more time than it takes to describe it.

In operating, Tait always aimed to do as little as possible. His incisions were short, never more than 2 or 2.5 inches unless a larger incision was necessary to remove a growth. His aim was to make the incision just large enough to admit his two large fingers. He said he learned this from Baker Brown. He opened the abdomen a little at one side of the median line and took care to avoid dividing the fibers of the rectus muscle. This practice he learned from A. McKenzie Edwards, one of his teachers at Edinburgh.

He was bitterly opposed to the use of the spray which at that time was in great vogue. I got the impression that his opposition to the spray and to antiseptic methods was chiefly based on his dislike of Lord Lister and Spencer Wells. He even refused to allow an application of antiseptics of any sort to the putrefying hysterectomy stumps which were in those days treated extraperitoneally. As a result, the atmosphere of his wards very often closely resembled that of a slaughterhouse. When one day I asked him to allow me to apply iodoform or carbolic acid to lessen the odor of decaying flesh, he curtly replied, "No," and added, "I cannot endure the smell of the stuff. I won't have it around." He did soon after begin the use of dry powdered boracic acid, insisting, however, that he used it only to keep the wound dry and not as an antiseptic.

Although Tait did not believe in antiseptics, he emphasized the necessity for cleanliness. This was perhaps his greatest contribu-

tion to surgery as he was really the father of surgical asepsis. He developed a technique which eliminated many of the perils of abdominal section and so materially reduced the mortality of this operation as to greatly enlarge its scope and enhance its usefulness. Men who followed his leadership in England, notably Greig Smith, Moynihan, and Mayo Robson, and in this country Joseph Price, Howard Kelly, and the Mayos, reduced the mortality rate to such a degree that the operation lost its terrors and soon came to head the list of major operations as a life saving procedure.

Though he opposed the Lister spray, Tait took the greatest care to keep his hands free from infection. If they became soiled at any time with an infectious fluid he refrained from operating for several days, having learned from experience that soap and water and even the use of the antiseptics then employed, would not always insure safety. Rubber gloves were of course not in use in those days. Instruments and ligatures were boiled. Sponges after being soaked over night in a one per cent carbolic acid solution were squeezed, put into a muslin bag, and hung up to dry. Only boiled water was used at operations.

At the time I was with him Mr. Tait boasted a record of 116 laparotomies with the same number of successive recoveries. The average mortality of the operation in this country at that time was, I believe, about 20 per cent. He attributed his success in ovariotomy to the adoption of Baker Brown's method of dropping the pedicle into the peritoneal cavity instead of treating it externally with the Spencer Wells clamp and introducing a drainage tube. Tait maintained that peritonitis was not likely to occur if the peritoneal cavity was kept dry.

Another reason for Tait's success was no doubt his radical and courageous departure from the long established method of dealing with the bowels. As late as 1883, Tait still practiced restriction of bowel activity after ovariotomy, insisting that the bowels should be confined for from 10 days to 2 weeks after operation. A little later, however, he made a radical change in his management of the bowels. Before the operation, the patient was thoroughly purged with saline laxatives and starved for 48 hours. After operation, the bowels instead of being confined were moved by enema on the second morning. Thorough evacuation of the colon on the second morning after operation was a dominant feature of the after-care of his patients. Drastic measures were used when necessary to secure an evacuation, and no food was given until after the bowels moved.

Tait would not administer anodynes of any sort so long as there was any hope of saving the patient. The patients sometimes suffered cruelly, but they rarely, if ever, received an anodyne drug of any sort unless they became moribund. He said, "I never give any drug unless the patient is going to die."

When asked what should be done in cases of peritonitis following abdominal section, he replied: "Nothing at all. The patient who has peritonitis after a surgical operation is certain to die. The time to cure peritonitis is before it begins. If the peritoneal cavity is kept well drained, peritonitis will not occur. The important thing is to keep the peritoneal cavity free from stagnant fluids. I am not afraid of germs. They cannot grow without food."

The carbolic acid spray of Lister was conscientiously employed by Spencer Wells and his followers, but Tait achieved better results without the spray than others did with it, employing otherwise the same technique. Undoubtedly, the abandonment of the Spencer Wells clamp and the use of the short sterile

ligature and the intraperitoneal treatment of the stump introduced by Baker Brown were the chief factors in reducing the mortality rate from the 25 per cent of Spencer Wells' first one thousand cases to less than 5 per cent in the hands of Tait, Bantock, Thornton, and Keith.

Tait's views were strongly supported by the doctrine of intestinal toxæmia which Bouchard had recently brought out. Widal, Roux, and other French investigators had recently shown that in certain conditions, particularly stasis, the pathogenic bacteria always found in the colon may become highly virulent and capable of invading the blood stream and the tissues and producing pleurisy, peritonitis, hepatic abscess, pyelitis, and other grave conditions. Roux had produced peritonitis and abscesses with pure cultures of bacillus coli. Tait maintained that these organisms could not develop without a liquid culture medium, and so he not only introduced a drain in every case, but took care to prevent accumulation of liquid in the abdominal cavity by applying suction to the drainage tube at frequent intervals so as to keep the abdominal cavity as dry as possible.

Tait's departure from the orthodox method of dealing with the bowels before and after laparotomy was doubtless one of his most important innovations. He led the way, however, in numerous departures from established methods and in undertaking new surgical procedures which have enormously increased the scope of abdominal surgery.

Tait claimed that he was the first to perform the operation for removal of the ovaries and tubes for the cure of chronic pelvic inflammation. He was first to operate for the removal of gall stones, first to operate in cases of ruptured tubal pregnancy, and the first to remove the uterine appendages for the relief of bleeding fibroids.

With his great intelligence and broad knowledge, Mr. Tait unfortunately gave no attention to personal hygiene. He was a good deal of a gourmand. He possessed an extraordinarily vigorous stomach which made no protest notwithstanding the enormous quantities of foods and wines as well as stronger liquors which he consumed at dinner. His gross eating habits were doubtless responsible for his premature death at the age of 54 after having previously submitted to an operation for removal of renal calculus.

His last medical paper was entitled "The History of a Sore Kidney," his own.

JOHN HARVEY KELLOGG.

MOVING PICTURES IN MEDICINE

TO one acquainted with the history of medicine, the rapid development of pedagogic principles in undergraduate teaching has been a source of great interest and pleasure. We have rapidly divorced ourselves from the amphitheater clinic and have brought the student into closer contact with the patient. While graduate teaching has lagged somewhat, yet, even here we are searching diligently for the means of imparting to those detached from medical centers the advances made in medical science, diagnosis, and treatment. This problem is a difficult one. In both groups the medical profession has eagerly seized upon any method that bids fair to increase the efficiency of teaching.

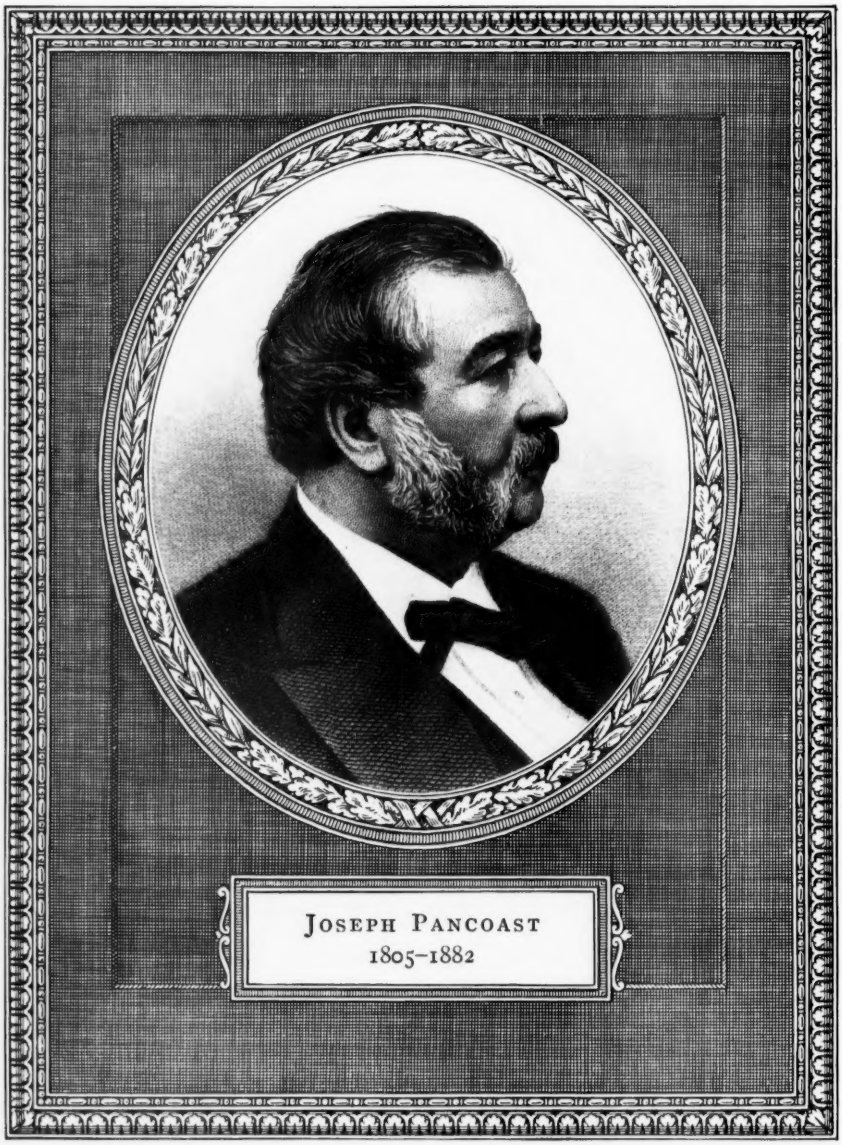
It was but natural that the moving pictures should be utilized for this purpose. It was also but natural that the early films should be those designed to depict some personal operative procedure produced in an inadequate way by the physician himself. Such pictures have their limitations, but were the logical first step.

In the last few years the thinking teacher has realized that the moving picture presents great possibilities in illustrating certain principles of medical science in which it is advisable to combine in a succinct form anatomical structure and function, embryological development, physiological processes, etc. This method of teaching can be utilized to bring to the practitioner of medicine the newer developments of medical science, newer procedures in diagnosis and treatment, the anatomy, physiology, graphic presentations of symptomotology, and principles of treatment. It presents them in a way that is readily understood and remembered by the busy practitioner.

This phase of teaching is in its infancy. The addition of the talking voice in certain cases will add to the clarity of the presentation and serve to emphasize certain principles. The moving picture as such offers a great possibility for the dissemination of scientific knowledge, particularly if those producing them keep constantly in mind the principles upon which such films should be constructed. The essential criteria should always be "Does the film teach sound, fundamental principles?"

At this stage, we should carefully avoid presenting controversial questions. If this be kept in mind and films are prepared to show the advances of medical science and are made available to the medical profession as a whole, it is probable that moving pictures will become one of the most valuable aids in the dissemination of medical knowledge. They can never replace the experience gained from personal contact with disease, nor can they supplant well established methods of teaching medicine; but as an adjunct to the present medical curriculum in schools and postgraduate study on the part of the practitioner, they should prove of inestimable value.

If such a method of medical instruction is to reach the highest level of efficiency, educational ideals and scientific accuracy must be maintained and photographic technique advanced constantly.



JOSEPH PANCOAST
1805–1882

MASTER SURGEONS OF AMERICA

JOSEPH PANCOAST

JOSEPH PANCOAST, the son of John and Anne (Abbott) Pancoast, was born near Burlington, New Jersey, November 23, 1805. Nothing is known of his early education. In 1828, he graduated from the medical department of the University of Pennsylvania and immediately began the practice of medicine in Philadelphia, specializing in surgery.

In 1830, the Philadelphia Association for Medical Instruction was formed. This was a quiz organization which consisted at first of Drs. Parish, Wood, S. G. Morton, John R. Barton, and Franklin Bache. Later Joseph Pancoast was connected with the organization. It was, however, short lived, for at the end of six years it disbanded.

The Philadelphia School of Anatomy was opened by Dr. James Valentine O'Brien Lawrance in 1820. He died in 1823 and the school passed into the hands of the gifted Dr. John D. Godman. In 1826 Godman went to Rutgers College as professor of anatomy and Dr. James Webster assumed charge of the school. Webster accepted in 1830 the chair of anatomy in the Geneva Medical College and in 1831 Joseph Pancoast, the fourth to take charge of this celebrated school, began his brilliant career as an anatomist and surgeon. On October 7, 1835, he was elected physician to the Philadelphia Hospital (Blockley), and soon after physician-in-chief to the Children's Hospital in the same Institution; from 1838 to 1845 he was one of the visiting surgeons to the same hospital. In 1838 he was called to the chair of surgery in the Jefferson Medical College, made vacant by the retirement of Dr. George McClellan, and gave up his charge of the Philadelphia School of Anatomy.

During the seven years he was connected with the school of anatomy he devoted much time to study and writing. In 1831, he translated Lobstein's *De nervi sympatheci humani fabrica et morbis*, Paris, 1823. This treatise contains an account of the first case of Addison's disease on record, though it was not recognized as a distinct disease until Addison published, in 1855, his classical work on the diseases of the suprarenal capsules (Henry). Later he edited Manec's *Great Sympathetic Nerve* and his *Cerebrospinal System in Man*, and fitly closed his career in the school of anatomy by editing, in 1838, a new edition of Wistar and Horner's *Anatomy*, to

which he added numerous notes, chiefly histological. This he still further remodeled in 1842 and again in 1846. For years this was the text used by the students at the Jefferson Medical College until it was supplanted by the excellent manual of Erasmus Wilson which eventually gave way to the familiar "Gray." In 1844 he published his *Treatise on Operative Surgery*, which passed through three editions; the third appearing in 1852. He contributed numerous articles to the *American Journal of the Medical Sciences*, *American Medical Intelligence*, *Medical Examiner*, besides publishing many papers on surgical and pathological subjects, introductory lectures, and, in 1856, his well known *Professional Glimpses Abroad*.

In 1841 Pancoast was transferred from the chair of surgery to that of anatomy which he resigned in 1874, after having filled for 36 years two of the most important chairs in the Jefferson Medical School: surgery and anatomy. In 1854 he was elected to the medical staff of the Pennsylvania Hospital and resigned in 1864. He was a member of his state, county, and city medical societies; the American Medical Association, Academy of Natural Sciences, College of Physicians of Philadelphia, and of the American Philosophical Society.

Surgery is indebted to Pancoast for a number of new operations. He devised the plow and groove, or plastic suture, by means of which four raw surfaces, the bevelled edges of the flap, and the margins of the groove cut by the side of the nose, to receive the flaps, come together. He used this suture in all his rhinoplastic operations with uniform success. He devised a fine needle, turned near the point into a hook, which he introduced just behind the cornea, through the anterior part of the vitreous humor; between the margin of the dilated iris and the lens. By means of this needle he was able to cut deeply the soft parts of the lens and withdraw along the line of entrance of the needle any hardened nucleus leaving the piece in the outer border of the vitreous humor. The operation was usually followed with little irritation.

For occlusion of the nasal duct in ordinary cases of epiphora he devised a small hollow ivory tube, from which the earthy matter had been removed, which he introduced from in front by a puncture of the lachrymal duct and left it to be slowly dissolved. In bad cases of internal strabismus he found that the tendon of the internal oblique muscle was often encircled by rigid connective tissue, and it was only by drawing the tendon out by means of a blunt hook and dividing the tendon that the strabismus could be corrected. In the case of large abscesses lying between the colon and cæcum and in front of the quadratus lumborum muscle, he performed successfully a lumbar operation. By cutting the posterior muscles of the velum palate and dividing any attachment they might have made to the pharynx, he several times restored a voice that had previously been unintelligible.

In empyema he raised a semicircular flap over the ribs, and, puncturing the pleura near the base of the flap, introduced a short catheter down to the inner

end of the puncture and secured it with a string, thus forming a fistulous opening with the movable flap serving as a valve when the catheter was removed. In 1862, he performed, for the first time, division of the trunks of the fifth pair of nerves as they emerge from their foramina, at the base of the skull, as a cure for tic douloureux. He devised an abdominal tourniquet, in 1860, which, by compressing the lower end of the aorta, shut off the arterial blood from the lower limbs, thus preventing death from loss of blood in amputations at the hip-joint or high up on the thigh. In cases of extroversion of the bladder he turned down cutaneous flaps from the abdomen and groin over the hollow raw surface of the open bladder. This operation was first performed by him in January, 1868.

"During the last fifteen years of his life writing had no charms for him, and when spoken to on the subject he said he thought he had done enough of that kind of work." Dr. Pancoast married, in 1829, Rebecca, daughter of Timothy Adams, of Philadelphia. He died March 7, 1882, in the seventy-seventh year of his age, "beloved and honored by all who knew him." WILLIAM SNOW MILLER.

THE SURGEON'S LIBRARY

OLD MASTERPIECES IN SURGERY

ALFRED BROWN, M.D., F.A.C.S., OMAHA

THE GYNECOLOGY OF MERCATUS

THE beginning of the sixteenth century found Spain rapidly gaining a rather prominent position in the medical world. There were, however, many reasons why this position should be more of a medical than a surgical one and a glance over the conditions in Spain at this time will explain them. Spain, through Columbus' discovery of America, had become one of the greatest maritime nations of the world, and also one of the richest. Sea service was that of peace and not of war. Her merchantmen sailed between Spain and the Indies, which were presumed to be off the coast of Asia, bent on missions of trade and commerce, and the days of the Armada bringing sea warfare with them did not come until late in the century. Physicians to care for the health of the sailors and treat them if epidemics arose were much more needed than surgeons to care for wounds, and because the doctors who followed the sea were being trained for the future duties required of them, surgery was largely neglected.

The great new country which was being opened up abounded in a flora that was new to the medical profession and the pharmacists. As the knowledge of physiology and general hygiene was very meager great faith was placed in medicines and the ample supply of new flowers and herbs coming in from the Indies afforded the investigative members of the medical profession material to experiment with in an effort to find new drugs for various diseases. For example, the medical department of the University of Alcalá which had been erected by Cardinal Jiminez de Cisneros, the head of the Inquisition, became greatly interested in this work, and its physicians devoted themselves almost wholly to botany until it became one of the foremost schools in Europe along this line. Relations with other European countries were quite friendly. In the latter years of the fifteenth century, the Moor had been driven out and so had the Jew, and those wars were over. With the adoption of her own Inquisition under Ferdinand, Spain had obtained the right to settle her own religious questions without consultation with Rome and had thus withdrawn from the European political and religious wars which were raging constantly. The Pyrenees made her more or less inaccessible. The rebellion of the Netherlands was accomplished without much fighting and the war with France, which had never been very active, was concluded in 1559. The necessity for army surgeons under these conditions was not very great, and the physicians were able to carry on the surgery necessary to peace time.

These factors helped to make surgery as a speciality unnecessary, but there was in addition a positive factor which rendered its study and practice more or less dangerous. Surgery, being founded upon anatomy, ran counter to the ideas of the Church, and Spain, having founded its own Inquisition which was hand in glove with the rulers, delegated to the Inquisitors the absolute power over its inhabitants in the matter of heresy. Anatomy was thus almost unknown. In Guadalupe there was a school where dissection was permitted by special privilege from the Pope but for the greater part it was neglected. The great Vesalius who came to Spain as personal physician to King Philip II was said to have been unable to find a single skull in all of Madrid.

Medical men of parts were, however, fairly numerous. One of the most learned of these physicians was Luis Mercado, more commonly known as Ludovicus Mercatus. He was born in Valladolid in 1520 and studied medicine at the University there. Later he became professor of medicine at his Alma Mater and in his old age was made emeritus. He likewise became physician to Philip II and after his death succeeded to the same position with the next King, Philip III. He died in 1606 at the age of 86 years. Mercatus wrote a surgery which was published in 1594 and a treatise on dislocations and fractures which appeared after his death in 1625. His most popular work, judging from the number of editions—seven—was his gynecology. It was first published in 1579 at Valladolid, then at Venice, Basle, Madrid, and Frankfort, and was included in the gynecological collection published in 1588.

The principal interest in this work centers in three chapters: the seventeenth chapter of the first book entitled "Concerning the Hard and Cancerous Tumors of the Breast," and eighteenth and nineteenth chapters of the second book "Concerning the Scirrhus Tumor of the Uterus" and "Concerning Cancer of the Uterus." In these he endeavors to point out the differentiation between benign and malignant tumors of these organs. His knowledge of the subject was considerable and is interesting as it shows a rather advanced point of view for this period.

DE MVLIERVM AFFECTIONIBVS; LIBRI IIII.

Primus, de Communibus Mulierum passionibus disserit.
Secundus, Virginum, & Viduarum morbos tractat.
Tertius, Sterilium & prægnantium } *accidentia ad vnguem*
Quartus, Puerperarum & Nutricum } *exequitur.*

LVDOVICO MERCATO MEDIC. DOCT.
Et in vallis Soletanę Academia primariæ Cathedræ
Professore Auctore.

CVM INDICE CAPITVM, TVM RERVM OMNIVM LOCVPLETISSIMO.

VENETIIS, Apud Felicem Valgrisium. M D LXXXVII.

REVIEWS OF NEW BOOKS

THOSE familiar with Dr. Foote's *Minor Surgery*[1] do not need to be told the excellence of the present volume. They will, however, be interested to know that the clinical sections have been preceded by a concise, complete description of the principles of surgical technique and the operations of minor surgery by Dr. Livingston, Dr. Foote's lately adopted collaborator; and that the senior author himself has preceded the sections on regional surgery with chapters on the general considerations of the different types of pathology—congenital defects and anomalies, wounds, fractures, inflammations, infections, tumors, etc., thus giving himself the opportunity of setting forth in one place the points applicable to the particular lesion wherever it may occur and freeing himself of the necessity of repeating them in each section.

With these additions the book emerges as an excellently arranged, complete treatise on the principles and practice of minor surgery. The sequence of sections—technique, bandaging, general consideration of the types of pathology, and finally the regional sections, in which are set forth the details of diagnosis and treatment, could not be improved upon. It insures completeness, avoids repetition, and facilitates reference.

For students the book is simple and fundamental; for practitioners and younger surgeons it provides easy reference and maintains the practical point of view. Whatever in bacteriology, pathology, or other theory is important in diagnosis or treatment is brought out in its proper place and its proper proportion. Treatment is described in sufficient detail so that it can be followed.

The style of both authors is excellent. One is not pained, as he is so often by medical texts, by grammatical inaccuracies and awkward diction. It is simple, terse, and epigrammatic. The elucidation of principles and the bringing home of points by quotations from the masters adds greatly to its effectiveness. J. R. H.

THE little book by Professor Naegeli[2] is an effort to teach an introduction to surgery chiefly by means of pictures. It is based upon the premise, which is stated by Professor Garré in the introduction, as follows:

"General experience and experimental psychology indicate that pictorial illustration is superior to any other didactic method for the purpose of retaining facts in the memory and turning them to practical use."

The reviewer quite agrees with this general principle but is disposed to criticize the manner in which Professor Naegeli has carried it out. If, as the author states, the book is to serve as an introduction to surgery, it should limit itself to elementary subjects and should not include reference to gunshot wounds of the intestine, strangulation of the bowel, rupture of the spleen, tuberculosis of the spine, surgery of malignant growths, biliary calculi, and so on.

While many of the diagrams are interesting and even striking, the treatment of the subject would seem to be almost too elementary for the medical student or practitioner but more suitable as an informational treatise to the laity. F. C.

MINOR surgery represents a very extensive and important field, yet there is little question but that in our didactic and clinical teaching of the undergraduate, too little attention is devoted to the "minor operation." Gastro-enterostomy or herniotomy intrigue the student or interne more than the treatment of a felon or the removal of a sebaceous cyst; yet repeatedly we encounter men, who, after an excellent interne service, are incompetent at such procedures.

The scheme evolved by Christopher in his *Minor Surgery*[3] is logical and interesting. It covers the scope of minor surgery after a method that parallels the plan of a well organized text of general surgery. The first seven chapters cover such general topics as wounds, foreign bodies, furuncles, carbuncles, burns, and gangrene. The remainder of the text is a regional minor surgery. Beginning with the head, injuries, infections, tumors, and deformities are considered. The same general scheme is used to cover neck, trunk, extremities, anus, rectum, and genitalia.

There are literally hosts of texts devoted to minor surgery, "Manuals," volumes on "surgical handicraft," etc. A large percentage of these are of little value. Either the author aims at brevity and compactness ruins his book, or he makes the fatal error of including too many procedures that may best be left to the domain of major surgery.

Christopher has given us an excellent book on minor surgery. It is beautifully written; the text is profusely and well illustrated. It should be of particular value to the man in a community remote from hospital centers where he cannot easily obtain advice and consultation on many of the so-called "minor," but important, surgical lesions.

J. R. BUCHBINDER.

RECENT years have found an ever increasing interest in matters of health. Physicians are anxiously striving to educate the people through

[1] PRINCIPLES AND PRACTICE OF MINOR SURGERY; A TEXTBOOK FOR STUDENTS AND PRACTITIONERS. By Edward Milton Foote, A.M., M.D., and Edward Meakin Livingston, B.Sc., M.D. 6th ed. New York and London: D. Appleton and Company, 1929.

[2] A GRAPHIC GUIDE TO ELEMENTARY SURGERY. By Prof. Dr. Th. Naegeli. Translated by J. Snowman, M.D., M.R.C.P. Introduction by Dr. C. Garré. New York: William Wood and Company, 1929.

[3] MINOR SURGERY. By Frederick Christopher, M.D., F.A.C.S. Foreword by Allen B. Kanavel, M.D., F.A.C.S. Philadelphia and London: W. B. Saunders Co., 1929.

books, magazines, and newspapers, while the latter, in turn, are always searching for material that they can understand. The facts concerning disease are hard to comprehend without some knowledge of the normal action of the human organs and the changes that take place in the organs as a result of disease.

Before reading such books as are written for the public concerning the various diseases, one would do well first to become familiar with the ways in which man in the past has recognized and struggled against the diseases that have caused him suffering and contributed to the shortening of his days. With such a background in the history of medicine, the nature of disease will become more clear, and the mental response to it more normal.

Medical histories, like medical textbooks, have been prepared a most exclusively for the use of physicians and others with scientific training. Now we have a book by Dr. Richard H. Hoffmann, called "The Struggle for Health,"[1] that has been written in clear, simple, and understandable language. The book is planned about the lives of the great men who have stood out because of their contributions and discoveries that have cheered and aided men in their struggle toward the goal of health.

Modern times find us making progress in the conquest of disease more rapidly than ever before. Our rapid strides now are, in large part, made possible by the work that has gone on before It is a duty, as well as a need, for all to acquaint themselves with the battle the great men who have gone before us have waged in the interests of mankind. And I know of a no more pleasant way of acquiring this knowledge than by recourse to the book by Doctor Hoffmann. H. B.

THE fifth volume[2] of the Oxford *Monographs on Diagnosis and Treatment* again covers its field in an authoritative, helpful manner. General methods of treatment including heliotherapy are accurately and explicitly described in a preliminary section. The X-ray picture of the normal chest is given in detail. The volume contains in addition over 150 skiagrams of pathological conditions of chests. These are large sized and are reproduced with remarkable faithfulness, thus making the work a valuable fund of information in regard to roentgen studies of the chest. Because of the author's close contact with the bronchoscopic work of Chevalier Jackson this form of diagnosis and treatment is stressed. Pneumonography by lipiodol injection is discussed. One-fourth of the work is devoted to pulmonary tuberculosis. The general subject of pulmonary accidents, parasitic diseases, and infections of the lung and pleura is thoroughly covered. This work is a valuable compendium for the practical man. PAUL STARR.

AS dogmatic with his opinions as he is prolific in his writings, Victor Pauchet[3] and his collaborators have given us an excellent résumé of their experience with the mooted treatment of gastric and duodenal ulcers. "Gastrectomy is, in principle, the only rational form of treatment for gastric and duodenal ulcers. Only through gastric resection wil the patient be cured and protected against relapses or late complications." This is Pauchet's opening statement, but he admits that there is much divergence of opinion because, as he truly states: "We do not yet know what an ulcer is, what is its nature, pathogenesis, or its immediate exciting etiology." He finds, clinically, that chronic colic stasis, appendicitis, cholecystitis, epiploitis, and inflammatory bands usually co-exist with gastric or duodenal ulcers. He also believes that all genuine ulcers exist in the presence of hyperchlorhydria and that nothing short of a sufficiently large gastrectomy to remove completely all of the pyloric glands will stop this hyperacidity. Pauchet summarizes his operative indications as follows: In duodenal ulcers: (1) with normal or hypo-acidity—gastro-enterostomy with invagination or excision of the ulcer; (2) with hyperacidity or hæmorrhage—duodeno-gastric or simple gastric resection. In gastric ulcers: (1) with hyperacidity—gastropyloric resection and gastro-enterostomy; (2) with normal or hypoacidity—gastropyloric resection in order to avoid possible secondary cancer.

The chapter on pre-operative care of the patient is detailed to the point of finicalness. How removal of tartar from the teeth or painting the gums with tincture of iodine can prevent pulmonary complications is hard to grasp! One cannot but applaud his physiologic statement that any pre-operative purgation should be discouraged. At present, 95 per cent of ulcer patients in Pauchet's clinic are operated upon under regional anæsthesia (abdominal wall and splanchnics). Simple excision of the ulcer is condemned and Balfour's cauterization is rarely used. The Péan (Billroth No. 1) technique is adopted in 15 per cent and the Pòlya in 85 per cent of his cases. The chapters devoted to perforating ulcers is excellent; postoperative complications and their treatment are fully covered. Chapter XII deals with Pauchet's operative statistics at St. Michel's hospital and analyses of 517 gastroduodenal operations with a total mortality of 8.7 per cent; in the past 2 years improved technique and better selection of the type of operation suitable to a given case has reduced the mortality to 6 per cent. The monograph is filled with valuable information and detailed methods of overcoming abnormal anatomical difficulties. G. DE TARNOWSKY.

[1] THE STRUGGLE FOR HEALTH. By Dr. Richard H. Hoffmann. New York: Horace Liveright, 1929.

[2] OXFORD MONOGRAPHS ON DIAGNOSIS AND TREATMENT. Edited by Henry A. Christian, M.D., Sc.D., LL.D. Vol. v. The Diagnosis and Treatment of Chronic Diseases of the Respiratory Tract; with Special Reference to the Lesions of the Trachea, Bronchi, Lungs, Pleura, etc. By Elmer H. Funk, M.D. New York: Oxford University Press, 1929.

[3] ULCÈRES DE L'ESTOMAC ET DU DUODÉNUM (ETUDE ANATOMO-CLINIQUE ET TRAITEMENT CHIRURGICAL). By Victor Pauchet, Gabriel Luquet, A. Hirchberg. Paris: Gaston Doin & Cie, 1929.

CLINICAL CONGRESS OF AMERICAN COLLEGE OF SURGEONS

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PLANNING FOR THE 1930 CLINICAL CONGRESS IN PHILADELPHIA

THE surgeons of Philadelphia, under the leadership of a strong and representative committee, are developing a highly attractive program of clinics and demonstrations for the entertainment of Fellows of the College and their guests at the twentieth annual Clinical Congress of the American College of Surgeons to be held in that city, October 13–17, 1930. All departments of surgery—general surgery, gynecology, obstetrics, orthopedics, urology, and surgery of the eye, ear, nose, throat and mouth—will be represented in this program.

Clinics and demonstrations will be given at the following hospitals: American Oncologic, American Stomach, Babies, Chestnut Hill, Children's, Children's Homeopathic, Cooper (Camden, N. J.), Evans Dental Institute, Episcopal, Frankford, Germantown, Graduate, Hahnemann, Jeanes, Jefferson, Jewish, Kensington, Lankenau, Methodist Episcopal, Misericordia, Mt. Sinai, Northeastern, Northwestern General, Orthopedic, Pennsylvania, Philadelphia General, Presbyterian, St. Agnes, St. Christopher's, St. Joseph's, St. Luke's, St. Mary's, Samaritan, Stetson, University, U. S. Naval, Wills Eye, Woman's Homeopathic, Woman's, Woman's Medical College, Woman's Southern Homeopathic. The clinical program will also include demonstrations in the laboratories of the medical schools: Jefferson Medical College, University of Pennsylvania, Temple University, Woman's Medical College.

Operative clinics and demonstrations in the hospitals are scheduled for Monday afternoon at 2 o'clock and for the mornings and afternoons of each of the following four days. A preliminary clinical program is to be published in the next issue of SURGERY, GYNECOLOGY AND OBSTETRICS.

The sub-committee in charge of the section on surgery of the eye, ear, nose and throat will arrange for a series of clinical demonstrations to be held at headquarters each morning, except Monday, in addition to the clinics in those specialties at the hospitals each afternoon.

Programs for a series of evening meetings are being prepared by the Executive Committee of the Congress. At the Presidential Meeting on Monday evening, the president-elect, Dr. C. Jeff Miller, of New Orleans, will be inaugurated and deliver the annual address. Another feature of that meeting will be the annual Murphy oration in surgery. For the scientific meetings on Tuesday, Wednesday and Thursday evenings, eminent surgeons of the United States and Canada with distinguished guests from abroad have been invited to present papers dealing with surgical subjects of present-day importance. At the annual convocation of the College on Friday evening, the 1930 class of candidates for fellowship in the College will be received.

The Congress opens at 10 o'clock on Monday morning with the annual hospital conference in the grand ballroom of the Bellevue-Stratford Hotel. An interesting program of papers, round table conferences and practical demonstrations dealing with problems related to hospital efficiency is being prepared. The hospital conference, which will continue on Tuesday and Wednesday, is planned to interest surgeons, hospital trustees,

executives and personnel generally, and an invitation to attend this conference is extended to all persons interested in the hospital field.

Other features of importance for which programs are now being prepared include a symposium on cancer and an all-day conference on traumatic surgery at which leaders in industry, education and labor together with representatives of insurance companies, surgeons and hospital administrators will participate.

General headquarters for the Congress will be established at the Bellevue-Stratford Hotel, corner of Broad and Walnut Streets, where the grand ballroom and other large rooms on the second floor, together with additional rooms on the roof have been reserved for the use of the Congress for scientific meetings, conferences, film exhibitions, registration and ticket bureaus, bulletin boards, executive offices, technical exhibition, etc.

In recent years a number of fine large hotels have been built in Philadelphia so that there are now ample first-class hotel facilities available for all who will attend.

An application for reduced railway fares on account of the meeting in Philadelphia is before the railway traffic associations, and it seems assured that a rate of one and one-half the regular first-class one-way fare will be in effect from all points in the United States and Canada.

LIMITED ATTENDANCE

Attendance at the Philadelphia session will be limited to a number that can be comfortably accommodated at the clinics, the limit of attendance being based upon the result of a survey of the amphitheaters, operating rooms, and laboratories in the hospitals and medical schools to determine their capacity for accommodating visitors. Under this plan it will be necessary for those who wish to attend to register in advance.

Attendance at all clinics and demonstrations will be controlled by means of special clinic tickets, which plan provides an efficient means for the distribution of the visiting surgeons among the several clinics, and insures against overcrowding, as the number of tickets issued for any clinic will be limited to the capacity of the room in which that clinic will be given.

A registration fee of $5.00 is required of each surgeon attending the annual Clinical Congress, such fees providing the funds with which to meet the expenses of the meeting. To each surgeon registering in advance a formal receipt for the registration fee is issued, which receipt is to be exchanged for a general admission card upon his registration at headquarters. This card, which is non-transferable, must be presented in order to secure clinic tickets and admission to the evening meetings.